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A REPORT TO THE NEW ENGLAND SURGICAL SOCIETY CONCERNING THE AMERICAN BOARD OF SURGERY*

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THE American Board of Surgery, established in 1937, has completed the first ten years of its activities. It seems appropriate that the present representative on the Board of the New England Surgical Society should report upon its accomplishments and the lessons learned during this time.

It is best, perhaps, to start with the reasons for its establishment. These were in general two: the chaotic situation regarding the qualifications that might reasonably be expected of a surgeon; and the inadequacy, both in quantity and in quality, of opportunities for obtaining the experience necessary to prepare one to meet these qualifications.

It was generally recognized at that time by those competent to judge that a large component of surgery was done by inadequately trained physicians devoting only a part of their time to this specialty. It was well nigh impossible to ascertain except by personal knowledge, not generally available and frequently unreliable, who was in fact a competent surgeon and who was not. In the *Directory of the American Medical Association* surgeons were authenticated on their own statements that they were practicing surgery and therefore presumably competent. In fact, it was generally known that a great many physicians in general practice were doing their own surgery even in communities where competent surgeons, confining their practice to surgery alone, were available.

This situation had been recognized and discussed for many years, and several solutions had been proposed and some attempted. Governmental licensure of specialists had been repeatedly suggested but was obviously impracticable if not undesirable, for this could only be accomplished by legislation in each of the states, a procedure that has not been satisfactory enough, when applied to the basic requirements for the practice of medicine in general, to encourage its application to the specialties. Membership in surgical societies was, with the exception of the surgical section of the American Medical Association and the American College of Surgeons, a mark of distinction too limited in its coverage. In the former one joined on signifying an interest in surgery, and in the latter the requirements were on a level pitched at initiating men actually doing surgery with the hope of improving their competency through postgraduate education.

The pattern of the boards for certification has become well established in other specialties, and that of surgery, a latecomer in the field, was set up along the same lines, being approved on the recommendation of the Advisory Board for Medical Specialties, by the Council on Medical Education and Hospitals of the American Medical Association. It should be made clear that, while recognized in this fashion, the American Board of Surgery was the result of the thought and action of many informed surgeons recognized by their colleagues as such, and approved by the major representative surgical associations; this has been evident in the continuing organization and functioning of the Board.

The objectives of the Board were and are to examine applicants and certify those meeting its requirements and — a purpose inextricably interwoven with this — to improve the opportunities for the training of surgeons by establishing the qualifications necessary for examination.

As in other specialty boards, the first certificates granted were to members of the founders' group "who had already amply demonstrated their fitness as trained specialists in surgery," 1152 surgeons being so certified. These were the senior surgeons to whom it would have been a hardship, if not an impossibility, to take the examinations. This method of admission could not be continued indefinitely without vitiating the purposes of the Board, and therefore it was announced at the start through suitable channels that the founders' group would be closed as of January 9, 1940. Unfortunately, for one reason or another, a number of sur-

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geons at the time presumably qualified, did not see fit to take advantage of this opportunity. Even more unfortunate, in spite of ample publicity concerning the requirements of the Board for examination, a group of younger men were unable or at least did not make any attempt to fit themselves to meet its basic requirements. To lower these requirements now to meet the desires of these men would be unfair to those who have spent the necessary time and energy to prepare themselves properly and would encourage others to attempt a similar short cut to certification.

Meanwhile, during the first ten years of activity of the Board, 1752 surgeons were certified by examination, making with the founders' group a total of 2904. As of January 1, 1948, there were 2062 and 3214, respectively, 310 having been certified by examination during the past year.

It is of interest to you, I am sure, to get some idea of the activity that is involved in this examination process. During the year 1946 the Board had about two thousand letters of inquiry. There were 789 completed applications, of which 40 per cent were at once acceptable for examination. Others may have become so with further information or preparation; the general experience is that only about 20 per cent of the completed applications are finally rejected. Out of some 700 men taking Part I of the examination, of whom 100 were repeaters, 25 per cent failed. Of 362 taking Part II approximately the same percentage also failed. It should be borne in mind that these data for Part II comprise numerous partial failures and that the actual number of final failures is somewhat lower because of success when the candidate repeats the examination.

The criteria that are used for the process are relatively simple, and at the same time reasonably rigorous. The candidate must demonstrate that he has a sound knowledge and understanding of the field of general surgery as of today. This is in part contained in the more detailed texts of general surgery, and in the surgical literature of the last ten years, which may not yet have arrived at this distinction. He must also give evidence that he is capable of employing this knowledge with judgment and skill in the care of the patient. The question always asked in summation is, "Is this potentially a safe and sound surgeon?" Such values are, of course, relative, as is the process of examination. Thus, it must be considered as a screening mechanism in which the sound and unsound grain, together with a considerable amount of chaff, go into the original hopper.

The review of the application is the first screen, and is concerned with whether the candidate has had a fair opportunity to learn through study and experience what the surgeon entering upon practice today should know, a matter requiring adequate exposure to both time and environment. This cannot be and is not rigidly defined and enforced, but one must bear in mind that no matter at what level the bar is set there will always be some who will insist upon attempting to crawl under it, even if the same amount of exertion expended properly would enable them to clear the obstacle without difficulty. The judgment of the Board has always been that a minimum of three years of institutional training is necessary, supplemented by two years of further experience. Recently, this has been modified upon the basis of experience indicating that one year of additional institutional training is usually equivalent to two years of supplementary experience gained otherwise. Therefore, four years of continuous, graded, institutional training is now recognized as adequate for admission to examination. It is the opinion that this represents the more desirable approach, but the number of opportunities for this intensive training are limited, and quite aside from the question of desirability it will be necessary for some time to continue also the requirements as established previously — namely, three years of institutional training with two years of supplementary program. There are thus two plans in effect by which a man can obtain the necessary preparation before taking the examination of the American Board of Surgery, one requiring five years, and the other four.

One may well ask whether the former is not quite satisfactory and what experience of the Board has led it to an adverse conclusion. The two supplementary years are almost as difficult to obtain as the additional year of institutional experience and often amount to little more than a waiting period, the educational value being only that which the prospective surgeon may vicariously gain in the first two years of practice. It is becoming increasingly difficult for him to obtain the hospital facilities necessary for surgical practice until he is at least eligible for examination. This fact, as well as the necessary requirement of the Board that he show evidence of further experience under suitable conditions, forces him into the hands of a preceptor. This relation may, when the preceptor is himself competent and also interested in the active and progressive training of the man, be highly desirable. Unfortunately, it is altogether too frequently the subject of abuse, and the young surgeon becomes the preceptor's assistant with inadequate remuneration and the experience that is acquired in doing the "scut" work for the older man. He may then find, having completed his period of service, that any attempt to establish himself in practice is blocked at every turn by the very man who has been his preceptor. I cannot conceive that any surgeon will in principle condone conscienceless exploitation, and certainly the American Board of Surgery cannot be a party to any such business. Yet, under present conditions and despite the exercise of the greatest possible caution, this situation may inadvertently arise in occasional cases. On the other hand the evaluation of institutions in this

respect — and they are by no means immune to the charge of exploitation of professional personnel at this level — can be more adequately and readily done, for they are fewer in number and more susceptible to investigation both in potentiality and in performance.

This screening of the applications is, then, the first step in the process for examination and a very necessary one, not only for the purposes of the Board but also for the best interests of many candidates. Some say that all who wish should be admitted to the examination without further ado. The result of this would be that either a large number of candidates would be failed or so many would pass that the Board would be discredited as an examining agency. To avoid the latter it would then tighten up its examination, failing some men who do not do well on the examinations, but have had good training, and passing others skilled in taking examinations, but who have had little or no experience. Less important perhaps is the number who would take a "flyer" at the examination on the chance of slipping through and would make it almost impossible to handle the load. Lastly, a major purpose of the Board, that of encouraging the development of adequate training programs, would be under these circumstances completely vitiated. This suggestion of relying completely upon examination is advanced by those who have not thought the question through or who suppose that this procedure is or can be made one of great accuracy. The methodology of examination has been thoroughly studied by experienced workers in the field of education and it has been well established that as the sole source of reference it has a considerable degree of error: approximately 20 per cent. The expression of results in precise mathematical terms gives an appearance of absolute and accurate value that is fictitious. It is at best only an approximation and must be reinforced in every way to reduce the error so far as possible; one of these is to evaluate the opportunity that the candidate has had for learning in the field of surgery.

This evaluation is made in the first instance by the secretary of the Board, whose long experience and sympathetic insight into the problems of the prospective surgeon are invaluable. In instances in which the problem is at all complicated, or in which his decision is questioned, the matter is carried before a special committee of the Board for further consideration and adjudication. Although the interests of the applicant concerned are viewed sympathetically and in detail, the paramount responsibility of the Board is to the public and its decisions must be so governed.

Another knotty problem concerned in the screening process is that of the ethical and moral standards of the candidate or, as I should prefer to put it, the pattern of behavior that may be expected of him in his future professional life. This has no reference to economic and social classification, race, creed, color or sex, because the pattern of behavior is not conditioned by these, per se, although as in all persons, adversity and misfortune may have a warping effect. It is the right of one in our form of civilization to be considered an individual rather than to be submerged in invidious categories. The record of the Board in this respect is one of which all surgeons may well be proud.

This "pattern of behavior" is unpredictable to a considerable degree even with adequate information. The usual letters of recommendation are perfunctory, sometimes even mendacious, and these sources of information are, on the whole, not reliable. They are worth obtaining, however, because of the occasional enlightening response, which may lead to further investigation. When there is any cause for suspicion certain persons whose reliability is beyond question are queried confidentially. This necessity may arise when a man has shifted about for no apparent reason. Without evidence to the contrary, it may be assumed that the behavior of a person who completes his training in one institution, or in several, with a sense of responsibility has been above reproach, at least up to that time. In spite of these precautions it is not only possible but probable that there are a certain number of certificated surgeons whose practice will be tagged "unethical." Occasionally this complaint comes to the Board, and it is taken most seriously. When the evidence justifies such a step, certification is recalled, but it is necessary to be certain that it will stand up in court, for it is likely that legal action for damages will be taken against the Board. In the majority of cases on detailed inquiry it is found that there is an internecine situation — one of those professional quarrels arising from jealousy, clash of personalities and so forth and giving rise to charges of "unethical conduct" that even locally are never taken so seriously that they are brought before a board of censors of the county or state society. Frequently it is a stab in the back of which the victim is unaware, for the informer insists on remaining anonymous. I dwell on this not to establish a thesis that the Board makes no errors in the evaluation of the future behavior of men accredited by it, but to call attention to the inherent difficulties in this problem. If the New England Surgical Society or any other organization can see its way clear to being of assistance to the Board in this matter it will be welcomed.

The examination having taken place, every effort is made to obtain a fair evaluation of the results. In the written examination (Part I) the papers of all failures or near failures are reviewed by two additional members after the initial reading by a member of the Board, and the mark primarily given confirmed or revalued. Papers that are still doubtful are brought before the Board as a whole and fully discussed with the additional information concern-

ing the candidate that may be pertinent, and the Secretary is directed regarding suitable action.

In the oral examination (Part II) held in clinical surgery on patients, in anatomy on the cadaver and in pathology on gross surgical specimens and microscopical sections carefully selected, approximately half the examiners are surgeons other than members of the Board and competent for this purpose. In clinical surgery teams are made up of a Board member and an accessory examiner who query together and separately grade the results. Since the candidate has two cases for examination, he is evaluated by two teams, a total of four surgeons, two of whom are Board members. This part of the examination is rated heavily, but there is also insistence that he demonstrate a knowledge of anatomy and pathology, adequate for sound surgery. This requires intensive study or review by most candidates, for it is the experience of the Board that men otherwise competent are frequently distressingly lacking in these subjects, which are fundamental to surgery.

The evaluations so obtained are assembled and gone over in a conference of all examiners in which questions and discussion are most welcome. With the failures and in doubtful cases, the information available in the secretary's files is brought to bear before a decision is reached, and then the Secretary is instructed concerning the action to be taken. It should be noted that in this conference, which is the last and most important step leading to certification, while the members of the Board bear the responsibility, they are assisted by an equal number of surgeons who are not Board members. This has the advantage of relieving it of any implication of bias and bringing into its activities at the most significant point a considerable number of surgeons who not only contribute materially to its deliberations but also learn at first hand the way in which the Board approaches this problem of examination and certification.

The second purpose is the improvement of the training of surgeons who may wish to qualify for the certificate of the Board. As pointed out above, the first and, as has been confirmed by experience, the most important step was taken when the general plan of training was outlined. This was made sufficiently rigorous in the over-all requirement to ensure the necessary time and application for the learning of surgery, but elastic in detail so that the candidates' individual ability and opportunity would not be forcibly compressed into a standard form. Unfortunately, as educators are well aware, the majority of students "jell" into a pattern if there is the slightest opportunity to do so, and it is almost traditional, as well as being consistent with human nature, that some will attempt to find some way of "beating the game." There is too much of the idea that the awarding of a certificate makes a surgeon, a fallacious concept that is by no mean limited to the experience of the American Boards of Surgery. For these candidates, the requirements must be rigorous, and vigilance must be constantly employed to see that in one place or another the standards are not evaded. For others, and fortunately there are many of these whose primary and fixed interest is in becoming sound and competent surgeons, great elasticity is allowable. The touchstone is whether the primary interest lies in the certificate or in the antecedent training. This can be ascertained fairly readily in most instances by the plan that the candidate submits for review; if he does not submit such a plan to the Board, it must assume that he is content with the customary pattern and is not concerned with its being adapted to his particular desires or opportunities.

A problem that remains a distressing one is the lack of suitable institutions both in quality and quantity capable of providing the opportunity for learning to be a surgeon. This has always been apparent and was recognized in 1936 by the American College of Surgeons when the regents adopted a policy directed toward increasing suitable residencies. So far as physical facilities, proper administration and organization, number of patients and competent surgeons on the staff are concerned, there are plenty of hospitals that could and should undertake the responsibility for training surgeons. But there is more to it than this, and this centers about a willingness of the professional staff to modify certain prerogatives and to divert a modicum of time and energy to a program directed at training surgeons. There must be a continuity of effort and maintenance of its operation at a constant high level, which requires that one person—a surgeon—must have the responsibility and the authority for carrying this on. When there is already an authoritative chief surgeon, he must in addition be competent, familiar with the literature, soundly educated and have a great interest in teaching in the broadest sense. To do this, he and the staff will have to sacrifice some of their customary activities; however, if a sound residency system is established, any concessions made by the staff will be more than compensated for by the assistance and stimulation of these younger men. Lastly, there comes a time when direct responsibility for the complete care, including operative procedures of an adequate number of patients, under supervision, of course, provides the final step in training. This seems to involve the greatest difficulty in the organization of the complete training program, particularly today when private patients are consuming bed capacity, leaving but few in the ward category. If one thinks of the resident at this stage of his development, at least four years out of medical school and with three years of surgical training, as a "junior attending" surgeon who is incidentally living in the hospital, the transition is less difficult.

In many hospitals connected with the schools of medicine such residency-training programs

have been established. These are particularly important as "pilot plants" for working out and demonstrating the proper approach. There are, however, by no means enough of these hospitals to train an adequate number of surgeons to replace those daily dropping by the wayside. Unfortunately, the idea has developed that without the facilities of a school of medicine it is impossible to carry out such training programs. This is not the case, and if the objectives projected in the formation of the Board of Surgery are to be achieved, many hospitals that are potentially capable of undertaking this training must do so. If the hospital is situated so that it is possible to do this under the guidance and with the assistance of a school of medicine, it may be the more readily accomplished, but this is not essential. There are now a sufficient number of younger surgeons who have been through this process of examination and training and who are therefore familiar with the concept and the application of it, so that given the opportunity this conversion of many hospitals quite away from schools of medicine and institutions for formal graduate instruction can be accomplished.

There are some who question the need or appropriateness of this method for the training of surgeons. To some it may seem an "academic" approach directed at the provision of the future teacher, rather than a "practical" method for obtaining practicing surgeons. If this is true, it has failed completely, for much more is required of the professional teacher than is provided in this program. More time and greater breadth of experience, particularly in the technics of teaching and investigation, are necessary for him. The requirements of the present training program, I must repeat, are directed at training competent, sound surgeons.

Another question that may be proposed is, Has the Board been reasonably successful in attaining its objectives? I think that there can be no question of the answer. The degree of recognition that has been accorded its diplomates has even proved embarrassing. The armed forces and the Veterans Administration, without the previous knowledge of the boards, recognized the qualifications of those physicians and surgeons who were diplomates, not only of the American Board of Surgery but of all the other specialty boards. That they were justified in so doing was proved by the experience during the war, for when these men were given appropriate assignments they performed superbly. In the Veterans Administration at the moment they are the core and foundation of the hope for improved professional care. Again, I wish to emphasize that the preferential position that these diplomates hold is not by any action of the boards, but is a voluntary and unsolicited recognition of their superior qualifications.

A similar situation for which the Board is not responsible is the special recognition of the diplomates accorded by many hospitals. This is not the concern of the American Board of Surgery except so far as the indiscreet, injudicious and arbitrary action of many of these institutions has served to throw discredit upon it. It has officially and in all honesty disclaimed any responsibility for this. Many hospitals have made a very laudable effort to elevate the level of professional services and have considered a blanket and complete restriction of facilities to certified specialists the simplest and most effective way of doing so. They have overlooked in many cases the fact that such an abrupt transition cannot be made. There may be on their staffs men who are known to be incompetent and whom they justifiably wish to dispossess. The easiest way, instead of deciding and acting in the individual case, is a blanket ruling requiring certification for staff membership and thus relieving themselves of the personal antagonism aroused by direct action. Unfortunately, this also dispossesses many surgeons who in their services have long since proved their competence, but who because of age fall in the intermediate group caught in this transitional period without certification. In doing this, the hospital "throws out the baby with the bath." In all fairness, although the ultimate transition in many hospitals may well end in making their facilities available for the most part only to those certified, such a regulation is at present premature. In fact, in some situations it may be a long time before this can be done, for there is still an inadequate number of diplomates to fill the needs, and in many of the smaller communities it would be an economic impossibility to support these specialists were they available. The objective, although it may be admirable, cannot be reached at once and in all cases, but must be approached by a period of transition and adjustment.

It is my impression, and that of many others, I am sure, that the success of the Board's activities has been well beyond what the originators of it might reasonably have expected. It has by no stretch of the imagination resolved the problem of providing a high grade of surgery for all, and there still remains a very great and disgraceful amount of incompetent, dangerous and unnecessary surgery. Recently, at a meeting in New York of the American College of Surgeons, Dr. Harold L. Foss pointed out what all of us know but rarely confess. This has received considerable publicity and will intensify the pressure upon the hospitals for restricting the specialists within them to those who are diplomates of their respective boards. This is a problem that the members of the New England Surgical Society might well study, for it is to them that the public should look for a proper answer. The Board cannot do this; it can only assist in the carrying on of its specific functions. It is not in any sense responsible for the development of a postgraduate plan of education, or of surveying and making

recommendations to hospitals regarding the organization of their staffs. It is not a continuing organization. When the man receives his certificate he does not become a member of a group such as this, or the American College of Surgeons. There is a place, however, for the postgraduate continuing education of the surgeon, a function that surgical organizations may perform and, particularly, on a national scale, the American College of Surgeons is splendidly organized to carry out.

Quite aside from this, the New England Surgical Society can assist the American Board of Surgery in its objectives of improving the training of surgeons and of examining those who have had adequate training. Beyond this there are certain needs apparent from the previous discussion. The necessity for more hospitals that can give acceptable residency training is great, and the members of this society should take the initiative and responsibility in developing their institutions to this end. Information — candid and dependable — regarding both hospital staffs in respect to teaching programs and applicants who may apply for admission to the examination is sorely needed and could be provided in New England by the members of this society. Thoughtful criticisms and suggestions are always welcome to the Board and can be readily presented through representatives of the Society. Above all is needed the moral support of the members if the Board is believed to be doing reasonably well what the Society endorsed in the beginning and what its representatives have attempted to carry out in conformity with its wishes.

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THE RAPID INJECTION OF A SOLUTION OF AMINO ACIDS

A Note on Its Clinical Tolerance in Man*

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EXPERIENCE with the parenteral administration of protein hydrolysates of five varieties (four acid and one enzymatically hydrolyzed) has led us to establish certain criteria for an ideal solution. In the first place, it must be shown to be nutritionally adequate by maintaining weight and nitrogen balance in the normal person when used as a sole source of nitrogen, and, in addition, positive nitrogen balance, weight gain and regeneration of serum albumin should be observed in persons depleted of protein. Secondly, side effects — that is, pyrogenic reactions, nausea, vomiting, venous thrombosis and anorexia — should be minimal. Thirdly, the solution must be nonantigenic. Finally, only small amounts of the material injected should appear in the urine, even with rapid infusion. Although the available hydrolysates have been shown to be nutritionally adequate as measured by nitrogen balance, unpleasant side effects occur frequently enough to limit their clinical usefulness.

Observations of the clinical tolerance to a 10 per cent solution of amino acids§ prepared by the recombination of fractions of an acid hydrolysate of casein are reported below. The solution is essentially devoid of glutamic and aspartic acids, and supplemented with dl-tryptophane and glycine.[1, 2] The results of 342 injections in 62 subjects over the past year indicate that the present solution of amino acids is better tolerated in man than those previously employed. The nutritional adequacy of the material and the urinary loss following infusion will be reported subsequently.

MATERIALS AND METHODS

The product under investigation is a complete acid hydrolysate of casein, contains no peptides, is essentially devoid of the dicarboxylic amino acids (glutamic and aspartic) and is supplemented with dl-tryptophane and glycine so that it contains the eight amino acids essential for man.[3] Additional dl-methionine was added to the product employed in our early studies, but is no longer supplied since excess methionine in man does not contribute nutritional advantage to a protein containing adequate quantities of this amino acid.[4, 5] The solution of amino acids now regularly contains adequate phenylalanine, although some of the early lots contained an insufficient amount of this essential amino acid.[6] The product is now neutralized with lactic acid to a pH of 7.2 to 7.4, whereas hydrochloric acid was previously used. Observations of the clinical tolerance of the 10 per cent solution of amino acids in man reported

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§Developed and distributed by Merck and Company, Inc., Rahway, New Jersey, who supplied the material used in this investigation.

below include all lots used during the past twelve months, but the similarity of their composition, despite several minor changes, makes a statistical evaluation valid. In Table 1 the composition of a representative lot of the solution of amino acids with respect to the eight amino acids essential for man[3] and for arginine and histidine is shown and is compared with that of casein. The results are based on analyses by microbiologic assay.[7–9]

All subjects selected were carefully followed each day. Many of the infusions early in this study, as well as subsequent ones, were given to subjects on the metabolic ward for more detailed study. Since this product had previously been shown to be well tolerated in dogs at very rapid rates of infusion,[1, 2] it was planned to administer it at rates exceeding those possible with the commercial protein hydrolysates that had previously been employed. In no case was the rate of infusion slowed to avoid or diminish a reaction. The following data were obtained: the diagnosis and body weight; quantity of material infused; quantity of other substances (glucose, vitamins and so forth) simultaneously infused; time required for infusion; and all untoward reactions, both immediate and delayed, no matter how mild or transient.

TABLE 1. *Comparison of the Composition of the 10 Per Cent Solution of Amino Acids with Casein.*

AMINO ACID	AMINO ACID SOLUTION* (L-FORM)	CASEIN †	CASEIN ‡	CASEIN §
	gm. per liter of 10% solution	*gm. per 100 gm.*		
Isoleucine	9.2	5.6	6.4	8.6
Leucine	17.7	9.9	9.9	10.5
Lysine	9.9	7.7	8.3	6.7
Methionine	4.0	2.6	2.6	3.1
Phenylalanine	4.2	5.9	5.2	4.8
Threonine	2.0	4.2	4.2	4.6
Tryptophane	0.9	1.1	1.2	1.4
Valine	7.2	6.7	6.2	5.8
Arginine	2.5	3.9	3.6	3.7
Histidine	4.5	2.8	2.6	3.0

*Also contains 0.9 gm. of d-tryptophane, 22.6 gm. of glycine, 0.2–0.5 gm. of glutamic acid, less than 0.05 gm. of aspartic acid and 0.2 gm. of tyrosine per liter of 10 per cent solution (microbiological assay).[7]

†Stokes et al.[7]

‡Hodson and Krueger.[8]

§Pearce et al.[9]

RESULTS

Classification of Subjects

The subjects were classified by diagnosis (Table 2). We were able to observe the most minute untoward effects in the normal volunteers (medical students and interns) even at extremely rapid rates of infusion (50 gm. of amino acids injected intravenously in ten minutes). Similar data were obtained from the convalescent patients. All other subjects were infused for therapeutic reasons as well as for data concerning tolerance. Most of the surgical patients received this product postoperatively as the sole source of protein, as did the newborn infants with infectious diarrhea. The patients with malnutrition and liver disease usually received the amino acid solution as a parenteral protein supplement. Many of the patients were critically ill.

TABLE 2. *Diagnostic Classification of Subjects.*

CLASSIFICATION		NO. OF SUBJECTS
Normal volunteers		11
Convalescent medical patients		5
Postoperative surgical patients		10
Partial gastrectomy	6	
Esophagectomy	2	
Bowel resection	2	
Malnutrition		16
Obstructive lesions of gastrointestinal tract	8	
Chronic medical illnesses	8	
Liver disease		13
Cirrhosis, active, severe	11	
Acute yellow atrophy	1	
Weil's disease	1	
Infectious diarrhea of newborn		7
Total		62

Quantity of Material Administered

The quantity of the 10 per cent solution of amino acids administered with each injection is shown in Table 3. Dosages of 30 cc. (3 gm. of amino acids) of the solution were given twice daily with an equal volume of 10 per cent dextrose to newborn infants with infectious diarrhea. This actually amounts to over 0.5 gm. of protein per kilogram of body weight at each injection and is roughly comparable to the injection of 500 cc. (50 gm. of amino acids) to a 70-kg. adult subject. All the 342 injections in this study were given in therapeutic amounts — that is, they supplied at least 0.5 gm. of protein per kilogram of body weight, and many were larger.

TABLE 3. *Quantity of Amino Acid Solution Administered.*

QUANTITY	NO. OF INJECTIONS
cc.	
30 (3 gm.)	90
500 (50 gm.)	162
1000 (100 gm.)	88
1500 (150 gm.)	2
Total	342

Composition of Material Administered

When administered for therapeutic purposes on the hospital wards, particularly when employed as the sole source of protein nutrient, the 10 per cent solution of amino acids was frequently given with 5 to 15 per cent dextrose in water or physi-

ologic saline solution. Vitamins prepared for parenteral use (B complex, C and K) were often added. Many of the malnourished group and patients with cirrhosis of the liver received 10 to 20 cc. of crude liver extract* in the infusion mixture. Sodium lactate was occasionally added. On three occasions the solution of amino acids (50 gm.) was given together with 75 gm. of human serum albumin (300 cc. of a 25 per cent solution). Although 240 of the 342 injections were "mixed," there was no apparent difference in the clinical tolerance whether the solution of amino acids was given alone or with these other substances. The only noticeable effect of the addition of other substances to the solution of amino acids was the decrease in the infusion rate necessitated by the increased fluid volume.

TABLE 4. *Number of Injections per Subject.*

No. of Injections	No. of Subjects
1	19
2–5	24
6–10	6
11–15	7
16 or more*	6
Total	62

*Two subjects received 16 injections, 1 received 18, 1 received 19, 1 received 20, and 1 received 21.

Number of Injections per Subject

Although many of the subjects received multiple injections (Table 4), no apparent increased incidence of untoward reactions was observed with subsequent infusions.

Rate of Infusion

In Table 5 are presented the rates of infusion of the solution of amino acids employed during this study. Only 7 per cent of all injections were given slowly, and most of these were in patients receiving the solution diluted to a large volume administered by constant intravenous drip. Over 70 per cent of the 342 infusions administered to our 62 subjects were given rapidly — that is, at rates corresponding to the infusion of 50 gm. of amino acids to a 70-kg. subject in less than an hour. Seventy-one per cent of these 242 rapid infusions were given at the rate of 50 gm. of amino acids to a 70-kg. subject in thirty minutes or less, and 44 per cent similarly in a quarter of an hour or less. These rates of infusion were far more rapid than could be attained with any of the protein hydrolysates previously employed, and at least four times faster than is usually recommended that they be given — that is, 50 gm. of amino acids to a 70-kg. adult in two hours.

*"Intraheptol," furnished by Lederle Laboratories, Inc., Pearl River, New York.

Reactions

The incidence of reactions at varying rates of infusion is presented in Table 5, and that of reactions occurring one or more times at all rates of infusion in Table 6.

The solution is pyrogen free. The single case of a pyrogenic reaction observed is no more than we should have anticipated from the infusion bottles and sets employed.

Nausea was observed in 9.4 per cent of all injections, and did not greatly vary in incidence with the rates of infusion. There does seem to be a greater incidence of nausea, however, the larger the quantity of material injected (Table 7). The increased incidence of nausea with larger infusions

TABLE 5. *Rate of Infusion and Incidence of Reactions of the 10 Per Cent Solution of Amino Acids.*

Rate of Administration	Corresponding Time Required for Injection*	No. of Injections	Thrombosis of Veins		Anorexia		Nausea		Vomiting		Frequency of Nausea and Vomiting from Protein Hydrolysates Previously Employed
			No.†	Percentage‡	No.†	Percentage‡	No.†	Percentage‡	No.†	Percentage‡	
gm./kg. / hr.	*hr.*										
<0.36	>2	23	0	—	1	4.3	2	8.7	1	4.3	Infrequent
0.36–0.71	1–2	77	2	2.6	2	2.6	5	6.5	1	1.3	Frequent
>0.71	<1	242	6	2.5	28	11.6	25	10.3	7	2.9	Very frequent
<0.35–>0.71	<1–>2	342	8	2.3	31	9.1	32	9.4	9	2.6	Administration usually impossible

*50 gm. of amino acids injected in a 70-kg. subject.
†Cases of reaction for each rate period.
‡Percentage of reaction for each rate period.

TABLE 6. *Reactions Occurring One or More Times in All Subjects at All Rates of Infusion.*

No. of Subjects	Thrombosis of Veins		Anorexia		Nausea		Vomiting	
	No.	Percentage	No.	Percentage	No.	Percentage	No.	Percentage
62	4	6.5	7	11.3	17	27.4	5	8.1

was more apparent in individual cases. Some subjects tolerated 50-gm. amounts but became nauseated with 100 gm., even at a slower rate of infusion. Although nausea was noted in 27.4 per cent of the subjects at one time or another during the period of therapy, it occurred in but 9.4 per cent of all injections. Of the 17 subjects who became nauseated during this study, 9 (53 per cent) accounted for 75 per cent, and 3 (18 per cent) for 37.5 per cent of all cases of nausea observed. It appears, therefore, that the nausea that followed this solution of amino acids was not definitely related to the speed of the infusion (and thus the height of the blood amino acid level) but, to some degree, to the quantity of material infused (apparently a "saturation" phenomenon), and, most of all, it appeared to be an individual reaction most frequently observed in a few subjects.

Since no infusions were slowed down to lessen reactions, the severity of the nausea can be judged by the end point of vomiting. In only 2.6 per cent of all infusions did vomiting occur. In 4 of the 9 cases it followed an attempt at forced feeding, and in another it followed the ingestion of brewer's yeast immediately after the infusion.

The true incidence of anorexia is probably higher than the 9.1 per cent observed, since this phenomenon was not always inquired about and some patients were tube fed or too ill to be aware of such a subtle subjective reaction. It is our impression, arrived at mainly from normal subjects, that a transient diminution of appetite usually occurs but is of very short duration and rarely persists for more than one or two hours. Two subjects studied on our metabolic ward received the solution of amino acids parenterally for nineteen and twenty days, respectively, and consumed at least 3000 calories of food by mouth at regular meal hours. Thus, with the infusion speeds readily attained with this product, proper spacing of the infusions between feedings allows for normal food consumption at the regular mealtimes.

In approximately 7.6 per cent of all injections, and in 21.0 per cent of all subjects, a transient sense of "flushing" or "warmth" without temperature change was noted. There was no significant change in the blood pressure in several subjects in whom this observation was made. In 3 of the 62 subjects (4.1 per cent) and in 5 of the 342 injections (1.5 per cent) an urge to move the bowels immediately after the infusion was noted, occurring only with rapid rates of infusion. A few subjects complained of a mild frontal headache. On careful questioning, several subjects were aware of the ability to "taste" and to a lesser extent to "smell" the infusion immediately after it had entered the vein. This sensation was transient. A very few subjects noted mild formication of the extremities.

The veins of all subjects infused with this solution were carefully watched. No cases of venous spasm, as has been reported with amino acid infusions,[10] were noted even with the rapid rates of infusion. Of the 342 infusions, thickening of the vein well occurred in 8 (2.3 per cent). In 2 subjects the thrombotic process appeared to be inflammatory, the redness appearing about one day after the infusion and subsiding after about twenty-four hours. In these cases there was complete occlusion of the vein. Although glucose (even in hypertonic solutions) was frequently given with the 10 per cent solution of amino acids, no increased incidence of thrombosis could be attributed to its use. Many subjects received multiple injections of this solution in the same vein, and thrombotic processes developed in only 4 of the entire group.

TABLE 7. *Relation between Incidence of Nausea and Quantity of Material Administered.*

Quantity Administered in Each Injection	No. of Injections	Cases of Nausea	
		No.	Percentage
cc.			
30 (3 gm.)*	90	?	?
500 (50 gm.)	162	17	10.5
1000 (100 gm.)	88	13	14.8
1500 (150 gm.)	2	2	100.0
Totals	342	32	
Average			9.4

*No objective reactions were observed in the 90 injections in the newborn infants with infectious diarrhea.

When a diuresis occurred it could be related to the quantity of fluid infused. One patient with rheumatic heart disease with mitral stenosis and another patient with multiple pulmonary and mediastinal metastases from an osteogenic sarcoma became short of breath during the infusion, but to no greater degree than when an equivalent quantity of fluid containing dextrose and water was given. Obviously one must employ discretion whenever solutions are administered parenterally to patients with diminished cardiac reserve. No anaphylactic reactions of any type were noted — and, indeed, none were anticipated since the product is a complete hydrolysate and does not contain peptides. Clinical acidosis was not apparent, even on repeated daily injections. No untoward reactions were observed in the 90 injections in 7 infants with infectious diarrhea of the newborn. Finally, no serious reactions or deaths due to these 342 injections in 62 subjects were observed.

Discussion

The 10 per cent solution of amino acids employed in this study had previously been shown by others to maintain weight and nitrogen balance in normal dogs[11] and to achieve positive nitrogen balance with regeneration of hemoglobin and plasma proteins in the doubly depleted Whipple dog.[12] The results obtained in the present study of the clinical

tolerance to rapid intravenous infusions of the solution of amino acids indicate that the present solution is better tolerated in man than those previously employed in this clinic. On several occasions the present product was satisfactorily employed, even though infused more rapidly, in persons in whom other hydrolysates had been discontinued because of nausea, vomiting or venous thrombosis. Over 70 per cent of the infusions were given at rates of administration more rapid than is usually possible with the available commercial hydrolysates. Because of the length of time previously required to infuse 50 to 100 gm. of amino acids and the frequency of nausea and vomiting, parenteral protein therapy has unfortunately been limited, because of both inconvenience and discomfort to the patient and the constant vigilance required by the physician. Since these disadvantages are minimized by the present product, parenteral protein hydrolysate therapy is more practical, and the physician may, if necessary, provide optimum protein feeding entirely by the intravenous route.

When employed as a parenteral protein supplement, the available protein hydrolysates may greatly diminish the voluntary oral food consumption, their apparent benefits being thus negated.[13] The present solution of amino acids appears to have marked superiority in this respect. Because of the rapidity of infusion attainable as well as the short duration of the anorexia that may ensue, the proper spacing of the injection between feedings allows for normal food consumption at regular mealtimes.

Although mixtures of synthetic amino acids are extremely well tolerated in man at rapid rates of infusion,[10, 14] have no significant effect on the oral food consumption[13] and can maintain nitrogen balance,[10, 14] their practical use is greatly limited because of both their excessive cost and the inefficiency due to the nonnutritive value and urinary loss of many of the unnatural amino acid isomers that they contain. Their value has been mainly as an investigative tool from which knowledge concerning the amino acids required by man, the tolerance of individual and mixtures of amino acids, the loss of these substances in the urine and the utilization of the unnatural isomer of the amino acids has been obtained.

An ideal product is not yet available. Although the dicarboxylic amino acids (glutamic and aspartic) contribute to the production of nausea and vomiting,[15, 16] their virtual removal from this product has not entirely eliminated these undesirable responses. The greater incidence of nausea when larger quantities of amino acids are infused (even at slower rates of infusion) suggests that a "saturation point" may be reached. Infusions of 50 gm. of amino acids twice daily (instead of a single 100-gm. infusion) might be expected to diminish reactions. As mentioned above, many cases of nausea appeared to be due to a lowered individual tolerance.

The relative freedom from venous thrombosis with the enzymatic casein hydrolysate previously employed ("amigen") is comparable to that of the present product, whereas most other hydrolysates caused a much higher incidence.

The solution of amino acids was well tolerated by the patients with the various illnesses reported in this paper, including those with severe active cirrhosis of the liver, and infants with infectious diarrhea of the newborn.

Summary

A 10 per cent solution of amino acids prepared by the complete acid hydrolysis of casein, devoid of peptides, largely freed of glutamic and aspartic acids and supplemented with dl-tryptophane and glycine, was administered 342 times to 62 subjects.

Over 70 per cent of all infusions were administered at rates usually unattainable with the previously available protein hydrolysates (50 gm. of amino acids in 70-kg. subject in less than an hour). All injections supplied at least 0.5 gm. of protein per kilogram of body weight, and many were larger.

The clinical tolerance of the solution of amino acids was evaluated in normal subjects as well as in patients with a variety of illnesses requiring parenteral protein therapy. There was no apparent difference in the clinical tolerance of the amino acid solution whether it was given with glucose, saline, lactate or vitamins, or when it was given repeatedly.

The incidence of pyrogenic reactions was 0.3 per cent; nausea 9.4 per cent; vomiting 2.6 per cent; venous thrombosis 2.3 per cent; and transient anorexia 9.1 per cent.

Because of the rapid rates of infusion attainable with but few untoward reactions, it is concluded that the solution of amino acids employed in this study was better tolerated in man than the protein hydrolysates previously used in this clinic.

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THE AGE DISTRIBUTION OF POLIOMYELITIS IN MASSACHUSETTS

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DATA concerning the incidence of poliomyelitis in Massachusetts have been collected by the Department of Public Health since 1907. This material had been studied statistically by Forsbeck and Luther[1] in 1930, and it was thought that a review of these older records, combined with the consideration of the age distribution. The material on the geographical aspects will be published elsewhere.

In the earlier study of Forsbeck and Luther,[1] it was noted that the percentage age distribution of the reported cases of poliomyelitis had been undergoing considerable variation. This change, data accumulated during the past fifteen years would yield additional information on this subject.

In the present analysis particular attention has been given to the age distribution and the geographical behavior of poliomyelitis in Massachusetts since 1907. This discussion is limited to a consideration of the age distribution. The material on the geographical aspects will be published elsewhere.

TABLE 1. *Age Distribution of Poliomyelitis in Massachusetts (1907–1945).*

YEAR	PATIENTS 0–4 YR. OF AGE		PATIENTS 5–9 YR. OF AGE		PATIENTS 10–14 YR. OF AGE		PATIENTS 15–19 YR. OF AGE		PATIENTS OVER 20 YR. OF AGE		KNOWN AGES	UNKNOWN AGES	ALL CASES
	NO.	PERCENTAGE	NO.	PERCENTAGE	NO.	PERCENTAGE	NO.	PERCENTAGE	NO.	PERCENTAGE	NO.	NO.	NO.
1907	—	—	—	—	—	—	—	—	—	—	—	234	234
1908	37	53.7	15	21.7	6	8.7	7	10.1	4	5.8	69	67	136
1909	408	66.3	115	18.7	44	7.2	15	2.4	33	5.4	615	308	923
1910	345	57.4	—	—	—	—	—	—	—	—	601	244	845
1911	—	(64.2)	—	—	—	—	—	—	—	—	—	260	260
1912	60	66.7	15	16.7	—	—	—	—	7	7.7	90	79	169
1913	181	62.0	58	19.9	—	—•	—	—	9	3.0	292	69	361
1914	62	64.6	15	15.6	—	—	—	—	9	9.4	96	55	151
1915	76	62.3	24	19.7	—	—	—	—	5	4.1	122	13	135
1916	1289	69.7	366	19.8	—	—	—	—	85	4.6	1850	77	1927
1917	—	(64.0)	—	(19.8)	—	—	—	—	—	(4.4)	—	174	174
1918	63	66.3	13	13.7	7	7.4	5	5.2	7	7.4	95	4	99
1919	39	61.9	13	20.6	6	9.5	3	4.8	2	3.2	63	3	66
1920	347	53.2	165	25.3	67	10.3	34	5.2	39	6.0	652	44	696
1921	98	43.6	53	23.5	30	13.3	13	5.8	31	13.8	225	8	233
1922	103	49.8	52	25.1	27	13.0	15	7.3	10	4.8	207	10	217
1923	126	59.4	46	21.7	20	9.4	8	3.8	12	5.7	212	11	223
1924	126	47.0	65	24.2	39	14.6	15	5.6	23	8.6	268	9	277
1925	81	50.3	27	16.8	28	17.4	8	5.0	17	10.5	161	6	167
1926	107	44.8	70	29.3	23	9.6	15	6.3	24	10.0	239	6	245
1927	536	45.9	361	30.9	133	11.4	70	6.0	67	5.8	1167	22	1189
1928	160	36.9	137	31.6	70	16.1	30	6.9	37	8.5	434	—	434
1929	32	27.8	38	33.1	19	16.5	15	13.0	11	9.6	115	4	119
1930	191	38.0	197	39.1	63	12.5	24	4.8	28	5.6	503	—	503
1931	398	27.9	579	40.5	230	16.1	113	7.9	108	7.6	1428	—	1428
1932	15	24.7	19	31.1	13	21.3	8	13.1	6	9.8	61	—	61
1933	98	27.8	131	37.1	61	17.3	24	6.8	39	11.0	353	—	353
1934	15	19.7	26	34.3	15	19.7	11	14.5	9	11.8	76	—	76
1935	448	32.2	461	33.2	298	21.4	98	7.1	85	6.1	1390	—	1390
1936	13	25.5	6	11.8	12	23.5	16	31.4	4	7.8	51	—	51
1937	74	21.1	116	33.0	85	24.2	40	11.4	36	10.3	351	—	351
1938	8	44.4	3	16.7	4	22.2	2	11.1	1	5.6	18	—	18
1939	17	22.4	27	35.5	14	18.4	10	13.2	8	10.5	76	—	76
1940	9	20.0	15	33.3	12	26.7	8	17.8	1	2.2	45	—	45
1941	43	23.6	62	34.1	33	18.1	16	8.8	28	15.4	182	—	182
1942	7	19.4	15	41.7	4	11.1	2	5.6	8	22.2	36	—	36
1943	71	28.1	83	33.0	43	17.1	25	9.9	30	11.9	252	—	252
1944	62	14.5	166	39.0	115	27.0	39	9.2	44	10.3	426	10	436
1945	103	19.8	181	34.6	119	23.0	49	9.4	69	13.2	521	6	527

In the present analysis particular attention has been given to the age distribution and the geographical behavior of poliomyelitis in Massachusetts since 1907. This discussion is limited to a consideration of the age distribution. The material on the geographical aspects will be published elsewhere.

In the earlier study of Forsbeck and Luther,[1] it was noted that the percentage age distribution of the reported cases of poliomyelitis had been undergoing considerable variation. This change, first noted about 1918, was still active in 1930. The same gradual alteration had been noted in other states by various authors.[2] To our knowledge, no satisfactory explanation of this phenomenon has yet been presented.

The age distribution of poliomyelitis in Massachusetts for the period during which records are available (1910–1947) is given in Table 1. The figures for the years 1907, 1911 and 1917 are

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lacking, and those for 1910 to 1916 are not available in all age groups.

These data, presented graphically in Figure 1, differ from those used by Forsbeck and Luther[1] in that the figures for the various age groups are here shown as five-year moving medians. By this method trends become more apparent. It is noted that the decrease in the group of patients under four years of age from 1918 to 1920 seems somewhat exaggerated when compared with the actual percentages as given in Table 1.

The missing values for the years 1911 and 1917 have been obtained by interpolating a figure mid-

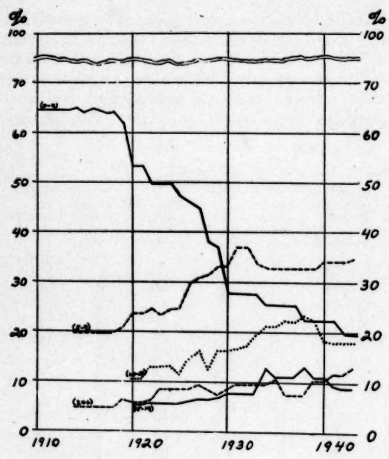

FIGURE 1. *Age Distribution of Poliomyelitis in Massachusetts, 1910-1943 (Five-Year Moving Medians).*

way between the middle values of the two years immediately preceding and following the year for which the value is lacking. The figures so obtained have been inserted in parentheses in Table 1.

Although the data are incomplete in the first decade (1910–1920), it is apparent that the age distribution during this period remained relatively constant. In the last two years of this period — namely, 1918 and 1919 — a decline occurred in the patients under four years of age, with a compensatory increase in the group from five to nine years of age. During the next decade (1920–1930) a continuation of this trend is noted, with the greatest decrease between 1927 and 1930 in the patients under four years of age. The compensating increases are most striking during this decade in the patients from five to nine and those from ten to fourteen years of age, with small increases in the two remaining categories. From 1930 to 1945 the decrease in the patients under four years of age is still apparent, although less marked, and the increases in other age groups are much less spectacular.

Changes in Age of the Population

In 1900, 1910 and 1920 persons under four years of age constituted respectively 10.1, 9.8 and 10.0 per cent of the total population in Massachusetts. This period of stability in the population corresponds with a period in which the age distribution of poliomyelitis remained stationary. In 1930 the same age group comprised 8.2 per cent of the entire population of the Commonwealth, and in 1940 it decreased by about a third — to 6.5 per cent. The decrease in the incidence of poliomyelitis in this age group, however, for the same period was over two thirds and therefore cannot be explained totally on the basis of aging population. The change in

TABLE 2. *Ratios of Rates under Five Years of Age to Total Rates for Paralytic and All Cases.*

YEAR	ALL PATIENTS			PARALYZED PATIENTS		
	UNDER 5	ALL AGES	RATIO	UNDER 5	ALL AGES	RATIO
1910	147.47	25.10	5.87	147.47	25.10	5.87
1920	96.00	18.07	5.31	96.00	18.07	5.31
1935	141.90	32.24	4.39	101.90	19.37	5.27
1940	3.19	1.04	3.06	2.13	0.39	5.41

population age distribution has undoubtedly been a significant factor, but that it is not the only factor is apparent from Table 2, from which the influence of the variation in annual incidence of poliomyelitis and the aging population has been removed by the use of a ratio between specific and total morbidity rates. From this table it is apparent that the ratio is lower for the more recent years (1935–1940) and this would not have occurred had the change in age distribution of the population been the only factor concerned.

During the period between 1930 and 1940 it was found as previously noted that the decrease in the incidence of poliomyelitis among children under four years of age was much less than that in the previous decades. The reduction is, however, proportionate to the decrease in that age group of the population during the same period and may therefore be considered as having been caused by it. However, the marked change that occurred between 1920 and 1930 must have been caused by some factor or factors in addition to that of alteration in age of the population. This factor would have to be one that was most marked between 1918 and 1930, particularly between 1927 and 1930, and one that remained active after 1930.

Reporting of Nonparalytic Poliomyelitis

The annual report of the Massachusetts Department of Public Health for 1909[3] states that

0.7 per cent of the cases of poliomyelitis reported during that year were nonparalytic. Data for certain other years also indicate that in general, nonparalytic cases constituted less than 1.0 per cent of the total number reported through 1917 and less than 5.0 per cent until 1920.

Reporting of nonparalytic cases became routine in 1935. During the succeeding eleven years nonparalytic cases have been recorded at an average rate of 37.0 per cent of the total for any one year. Partially complete data for 1931 show that 35.0 per cent of the cases were nonparalytic. There has been no apparent tendency for nonparalytic poliomyelitis either to increase or to decrease during the past decade. There is evidence, therefore, of an increase in the reporting of nonparalytic poliomyelitis from about 1.0 per cent to approximately 33.3 per cent of the total number of cases.

Since the rate of reporting of nonparalytic cases has been relatively steady since 1935, this increase in reported cases of nonparalytic poliomyelitis occurred in the period between 1918 and 1931. The significant factors responsible were greater knowledge of the disease as a result of the studies made during the great New York epidemic of 1916 and the introduction of convalescent serum for the treatment of preparalytic cases. The reporting of nonparalytic cases was also stimulated to a great extent by the difficulties encountered in differentiating preparalytic from nonparalytic cases. Apparently, once the habit of reporting such cases had been established, it continued to the present.

Age Distribution of Paralytic and Nonparalytic Poliomyelitis

Although paralytic and nonparalytic poliomyelitis in Massachusetts are alike so far as the seasonal, geographic and sex distribution is concerned, the age distribution of the two varieties is dissimilar. The greatest number of nonparalytic cases occurs in the patients five to nine years of age, the second greatest in those ten to fifteen years of age, and the least in those under four years of age, the latter being the group in which the greatest number of paralyzed cases are found. Difficulties in diagnosing nonparalytic poliomyelitis in infants and very young children may be the most significant factor in this observed difference. The age distribution of the nonparalytic cases has remained relatively unchanged since 1935 (Table 3). Although the data concerning nonparalytic cases in the years prior to 1935 are less complete, there is no evidence from the available information that the age distribution of this disease has been different for these periods.

Since the age distribution of the nonparalytic cases, as noted above, differs from that of the paralytic, it would be expected that the addition of a gradually increasing proportion of nonparalytic cases would alter considerably the age distribution of the total. The more marked changes would be a decrease in number of nonparalytic cases in the patients under four years of age and increases in those five to nine and ten to fourteen years of age. The change would be most striking between 1927

Table 3. *Percentage Age Distribution of Nonparalytic Poliomyelitis in Massachusetts (1935–1945).*

Age yr.	No. of Cases	Percentage
0–4	221	18.2
5–9	489	40.3
10–14	302	24.9
15–19	110	9.1
20	93	7.6
Total	1,215	

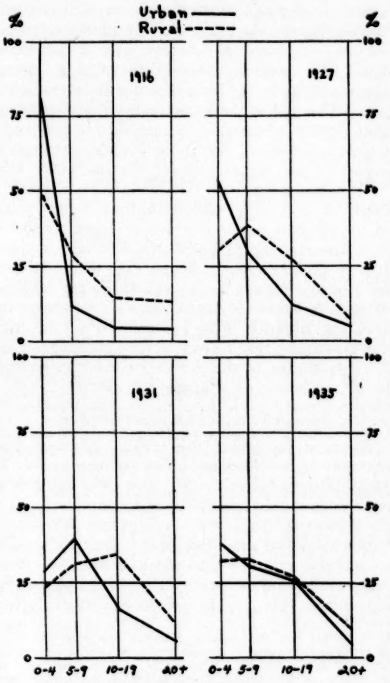

Figure 2. *Urban (Cities with Populations of More Than 100,000) and Rural (Towns with Populations of Less than 2500) Age Distribution of Poliomyelitis in Massachusetts for Epidemic Years.*

and 1931, when the stimulus to increased reporting of nonparalytic cases was most dominant. This is precisely the pattern of the change in the age distribution of the disease from 1918 to 1930 that was not wholly explained by the variation in the population as mentioned above.

If the change in age of the population and increased reporting of nonparalytic cases were responsible for the major portion of the alteration in percentage age distribution, and if only paralyzed cases were considered, the relation between the morbidity rates for poliomyelitis in the whole population and in the group under five years of age should show no marked change during the period under observation. In Table 2 such a relation is presented for sample years. Census years have been used except for 1935. This table suggests a relatively constant ratio of approximately 5 cases in the patients under five years of age, to each 1 in the general population for the four sample years. It may therefore be concluded that there has been no essential change in the epidemiologic behavior of poliomyelitis in Massachusetts and that the apparent variation in its age distribution has been due almost entirely to two factors: a change in the age distribution of the population, and an increase in the reporting of nonparalytic cases.

Urban-Rural Age Distribution

Figure 2 represents the urban and rural percentage age distribution of poliomyelitis in Massachusetts for epidemic years. The age grouping ten to nineteen only is used because the data for 1916 are available only in this form. The distribution shows the same changes previously discussed — namely, a decrease in the patients under four years of age and increases in the older age groups. In addition, the marked difference between urban and rural distributions that existed in 1916 has gradually decreased until in 1935, although still apparent, it is less striking. Similar differences between urban and rural age distributions are found in other communicable diseases, particularly the virus infections of childhood. It is generally accepted that these changes are due to the greater dispersion of the population in rural areas, resulting in relatively fewer cases at early ages and more among older persons. In Massachusetts there are comparatively few isolated communities at the present time, whereas in 1916 there was a larger number of truly rural areas. The great increase in automobile travel, the expansion of cities and changes in rural school systems have tended to increase the opportunities for exposure of rural populations to infectious diseases.

Summary

Changes in the percentage age distribution of poliomyelitis in Massachusetts since 1907 are presented.

It is concluded that there has been no essential change in the epidemiologic behavior of the disease, and that the apparent change in its age distribution has been caused by an alteration in the age of the population and an increase in the reporting of nonparalytic cases.

The difference between urban and rural age distributions of poliomyelitis in Massachusetts is rapidly disappearing.

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SPLENIC RUPTURE IN INFECTIOUS MONONUCLEOSIS*

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RUPTURE of the spleen in infectious mononucleosis is a rare complication of a rather common disease. King,[1] in 1941, presented the first authenticated case. Since then 13 additional cases have been reported.[2–8]

It is the purpose of this paper to present 2 additional cases of infectious mononucleosis complicated by splenic rupture. In 1 of these the primary pathologic process was unsuspected preoperatively. In the other the diagnosis had been established before operation was undertaken. Subsequent to splenectomy both patients recovered full health, and microscopical changes consistent with those described as occurring in infectious mononucleosis were found in both the removed spleens. In one of these cases rupture apparently followed slight trauma to the abdomen. In the other, spontaneous rupture appears to have occurred.

Previously reported cases include only two in which splenic rupture followed definite abdominal trauma.[5, 6] It must therefore be emphasized that rupture of the spleen in infectious mononucleosis may occur spontaneously or as the result of such trauma as that involved in defecation or in palpation of the abdomen in the course of physical examination.

Smith and Custer[6] first called attention to the time element of pre-existing symptoms in relation to splenic rupture. Table 1 includes an estimate of this time factor in the reported cases. In only 1 case (reported below) did splenic rupture occur less than two weeks after the onset of symptoms. This tends to support the statement made by Smith and Custer[6] that not until the third week have the capsular and trabecular changes progressed sufficiently to permit rupture, either spontaneously or as the result of slight trauma.

Consideration of the data summarized in Table 1 leads to the impression that a preoperative diagnosis of infectious mononucleosis was made in only 7 of the 16 cases. It is interesting to note that in the 7 cases in which a diagnosis of infectious mononucleosis had been made there was only 1 subsequent death; in that case no operation was performed because the diagnosis of ruptured spleen was not made. In the 9 undiagnosed cases 3 patients died without benefit of operation, and 2 died postoperatively. The over-all mortality in the 16 cases was 30 per cent. Although this series is too small to draw statistical conclusions it seems reasonable to hope that with increasing awareness of the fact that spontaneous rupture of the spleen may occur in infectious mononucleosis, more prompt and decisive operation will result, with subsequent improvement of the survival rate in this dangerous complication of an otherwise benign disease.

CASE REPORTS

Case 1. R. E. M., a 19-year-old seaman second class, entered the hospital on September 8, 1947, with the chief complaint of pain in the left upper abdomen of 48 hours' duration. One month before admission he had developed swollen cervical lymph nodes and fever associated with malaise and anorexia. He had no sore throat or infection about the head or neck at that time to account for the lymphadenopathy. He was treated with penicillin, and apparently his symptoms subsided in several days. On September 4, while roughhousing, he was struck a light blow in the epigastrium, which was not severe enough to knock him down. Upon arising on September 6 he noted mild left-upper-quadrant pain, which suddenly became severe and doubled him up. This pain remained constant until entry, except for periods of increased severity when it radiated to his left shoulder. His symptoms were aggravated by coughing or deep breathing and relieved partially when he sat upright. On the day of admission feverishness and generalized abdominal pain were present. Nausea, vomiting, diarrhea and headache were denied.

Physical examination revealed a hot, flushed, somewhat dehydrated patient; there was a questionable icteric tint to the scleras. There was limitation of expansion of the left side of the chest because of the abdominal pain, but the lungs were clear. Examination of the heart was not remarkable. The abdomen was diffusely tender, but most tender in the left upper quadrant, where there was a sense of fullness and a questionable mass. Throughout the abdomen there was "a doughy type of rigidity."

The temperature was 100°F., the pulse 108, and the blood pressure 124/72.

Examination of the blood disclosed a red-cell count of 3,400,000, with a hemoglobin of 9.5 gm., and a white-cell count of 7200. A differential white-cell count was not done at that time.

The patient was taken directly to the operating room. A high, left-rectus-muscle-splitting incision was made under nitrous oxide, oxygen and ether endotracheal anesthesia. Blood clots and free old blood were found in the peritoneal cavity. An enlarged spleen was encountered with a large hematoma on its lateral surface. The spleen was not adherent, but in freeing the superior pole by blunt dissection, it was further ruptured into several fragments. The fragments were manually removed, and the pedicle ligated en masse. During manipulation of the spleen the patient went into profound circulatory collapse, which responded to the intravenous infusion of 1500 cc. of whole blood. The abdominal wall was closed in layers without drainage.

The postoperative course was remarkably smooth, without any evidence of a pulmonary complication. He became ambulatory on the 3rd postoperative day.

On September 23 heterophil-antibody agglutination was positive in dilutions up to 1:1792, and a blood smear showed 63 per cent neutrophils, 34 per cent lymphocytes, 1 per cent

*The opinions or assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

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monocytes, 1 per cent basophils and 1 per cent eosinophils. A few young lymphocytes were noted.

The gross specimen consisted of a spleen weighing 444 gm. in the fixed state and of usual shape. The capsule was ruptured, and the surface was covered with clotted blood. The cut surface revealed many areas of hemorrhage and clotted blood within the spleen itself. These areas varied from pin-point size up to several centimeters in diameter. The splenic pulp appeared homogeneous and grayish. On microscopical examination, performed at the United States Naval Medical School, section of the spleen showed several areas of extensive hemorrhage, with disruption of the tissue in these areas. Elsewhere the splenic structure demonstrated a normal number of malpighian bodies. In many of them the germinal cells were enlarged and disclosed rather marked reticuloendothelial hyperplasia. Throughout, the red pulp and sinusoids were somewhat dilated, and the reticuloendothelial cells lining the sinusoids had markedly enlarged, somewhat irregular, oval to round nuclei, which were very vesicular. There was rather marked hyperplasia of these cells throughout the tissue. Many polymorphonuclear leukocytes were found scattered throughout the tissue. Many of the sinusoids contained rather bizarre atypical cells. The cellular detail was rather distorted, and a definitive diagnosis of infectious mononucleosis could not be made. However, the histologic appearance was compatible with such a diagnosis.

The pathological diagnosis was probable infectious mononucleosis, with rupture of the spleen.*

The diagnosis in this case was not suspected preoperatively. However, the history of lymphadenopathy, the strongly positive heterophil-antibody agglutination and the splenic disease made the diagnosis extremely probable.[9]

Case 2. C. K., a 19-year-old seaman first class, was admitted to the hospital on December 14, 1947, complaining of nausea and vomiting of 4 days' duration and abdominal pain of 24 hours' duration. The patient had been perfectly well until 1 week before admission, when he developed a "cold" characterized by coryza, a moderately severe sore throat, slight cough and malaise. He had no headache and did not feel sick enough to report to his doctor. Four days before admission he became nauseated and began vomiting. In the course of the next 4 days he vomited frequently and took only fluids by mouth. His bowel movements were normal. On the day prior to admission, while retching, he suddenly developed a severe, sharp pain in the left upper quadrant, which radiated to the left shoulder, and he reported to the sick bay. Shortly thereafter the pain became generalized. Physical examination at that time revealed a pale young man in moderately acute distress. The temperature was 99.4°F., the pulse 88, and the respirations 20. The pharynx was injected, and the tonsils enlarged and inflamed. The abdomen was soft and slightly distended, and there was generalized abdominal tenderness. Peristalsis was hyperactive. The patient was given intravenous fluids, Wangensteen suction and was placed under observation. On the following day extreme tenderness below the left costal margin and rebound tenderness in the hypochondrium were present, a questionable mass in the left upper quadrant was felt, and he was transferred to the hospital.

TABLE 1. *Data Concerning Rupture of the Spleen in Infectious Mononucleosis.*

AUTHOR	PREOPERATIVE DIAGNOSIS OF INFECTIOUS MONONUCLEOSIS	OUTCOME*	POSTOPERATIVE COMPLICATIONS	DURATION OF SYMPTOMS BEFORE SPLENIC RUPTURE
				days
King[1]	Doubtful	Recovery	None	17
Ziegler[2]	Made	Death	—†	Doubtful
Darley et al.[3]	Made	Recovery	None	15
Davis[4]	Made	Recovery	None	15
Milne[5]	Doubtful	Recovery	Atelectasis and pneumonia	Doubtful
Smith and Custer[6]				
Case 1	Not made	Death after operation	Pneumonia and pulmonary embolism	21
Case 2	Made	Recovery	None	15
Case 3	Not made	Death	—†	Doubtful
Case 4	Made	Recovery	None	18
Case 5	Made	Recovery	Pneumonia	Doubtful
Case 6	Not made	Death after operation	Pulmonary embolism	30
Case 7	Not made	Death	—†	14
Vaughan et al.[7]	Not made	Recovery	? pneumonia	30
Spence[8]	Not made	Recovery	Pleural effusion	Doubtful
Timmes, Averill and Metcalfe				
Case 1	Not made	Recovery	None	30
Case 2	Made	Recovery	None	7

*In all cases in which the outcome is reported as "recovery" splenectomy was performed.
†No operation performed.

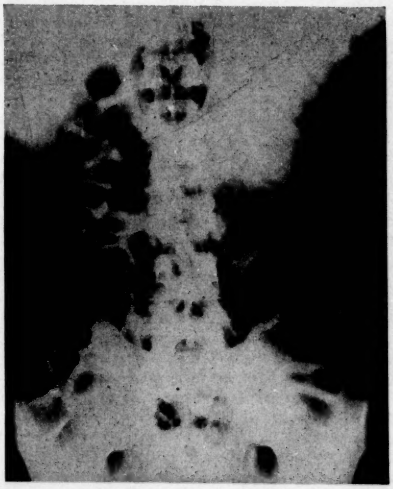

FIGURE 1. *Plain Film of the Abdomen in Case 2, Showing the Large Mass in the Left Upper Quadrant.*

*Dr. Charles F. Geschickter, who reviewed the slides, concurred in the diagnosis.

Physical examination revealed an acutely ill, toxic, dehydrated and hyperpneic patient. Bilateral enlargement of the axillary lymph nodes was noted, and a few coarse rhonchi were heard throughout both lung fields.

The temperature was 104°F., the pulse 112, and the respirations 26. The blood pressure was 125/80.

A plain film of the abdomen revealed gaseous distention of the small bowel and a large left-upper-quadrant mass (Fig. 1).

Examination of the blood disclosed a red-cell count of 4,100,000, with a hemoglobin of 12.5 gm., and a white-cell count of 9600, with 13 per cent neutrophils, 64 per cent lymphocytes and 3 per cent monocytes. Many of the lymphocytes were large and immature, and were very suggestive of infectious mononucleosis. A blood Kahn test was negative. Initially, examination of the urine showed the presence of acetone bodies, which subsequently cleared with rehydration.

A Miller–Abbott tube was passed. The patient was started on penicillin therapy, 100,000 units every 3 hours, and given intravenous fluids.

On December 15 a heterophil-antibody agglutination was positive in dilutions up to 1:3584. With persistence of abdominal findings and the probability of infectious mononucleosis, a tentative diagnosis of a subcapsular splenic hemorrhage was made.

Under nitrous oxide, oxygen and ether endotracheal anesthesia a left subcostal incision was made, with transverse sectioning of the left rectus muscle. When the peritoneal cavity was entered blood clots and free, dark blood were encountered, estimated at 300 cc. in amount. The spleen was enlarged and exhibited a ragged laceration of the capsule measuring 4 by 6 cm. on the anterior surface of the lower pole and a few small lacerations of the capsule over the lateral surface. The splenic pulp lay exposed through the capsular rents, did not appear ruptured and was not bleeding. As the spleen was not adherent it was easily separated by blunt dissection from its diaphragmatic and renal attachments and manipulated to a position outside the abdomen. Segmental ligation of the pedicle was carried out, and after all free blood had been aspirated from the peritoneal cavity the abdominal wall was closed in layers.

The postoperative course was essentially uneventful. The feared atelectasis, pneumonia and pleural effusion frequently seen after splenectomy were avoided, it is believed, by frequent inspiratory exercises, turning maneuvers, carbon dioxide inhalations and the prophylactic use of penicillin.

The gross specimen consisted of a spleen weighing 230 gm. when fixed. It was markedly abnormal, containing a large amount of clotted blood beneath the capsule, and appeared more friable than normal when handled. On microscopical examination the sections were those of a spleen with slight to moderate distortion of the architecture. The capsule was of average thickness throughout. The parenchyma was made up for the most part of large mononuclear endothelial-type cells, with round to oval vesicular nuclei. They were indistinguishable from the large, swollen endothelial cells lining the sinusoids. The malpighian corpuscles were moderately obliterated, with no distinguishable central arterioles. There were many subcapsular dilated sinusoids filled with red cells and atypical mononuclear leukocytes.

The pathological diagnoses were acute splenitis and infectious mononucleosis.

Discussion

Rupture of the spleen in infectious mononucleosis, although rare, does occur. Furthermore, its apparent rarity may be due in some part to lack of awareness of the syndrome among those who are called upon for emergency removal of the ruptured spleens. With more general appreciation of the fact that in infectious mononucleosis splenic rupture may occur either spontaneously or as the result of minimal abdominal trauma three things may be accomplished. The performance of diagnostic tests for infectious mononucleosis in patients suspected of splenic rupture, and in the postoperative study of those from whom a ruptured spleen has been removed, may lead to a revision of the present estimate of the rarity of the condition discussed. Secondly, such simple prophylactic measures as avoidance of heavy exertion during the phase of infectious mononucleosis in which the spleen is enlarged and avoidance of repeated palpation of an enlarged spleen in patients with infectious mononucleosis, as emphasized by Smith and Custer,[6] may lower the incidence of this complication (this consideration assumes increased importance when it is realized that although infectious mononucleosis is a benign disease splenic rupture carries a high mortality). Finally, careful observation of patients with infectious mononucleosis for evidence of abdominal pain or acute blood loss may lead to more prompt and definitive surgical action, with resultant improvement in the mortality figures of this complication. Awareness of the syndrome certainly played an important role in the decision to operate on the second case reported above when the diagnosis of infectious mononucleosis had been established.

In neither of the cases reported did the total white-cell count rise above 10,000 after splenic rupture. This is at variance with the usual findings in splenic rupture and with the majority of the reported cases of the syndrome under discussion.

Smith and Custer[6] were the first to state that rupture of the spleen seldom occurs prior to the third week in the course of infectious mononucleosis. A review of the literature appears to support this statement.

Summary

Fourteen cases of splenic rupture in infectious mononucleosis reported in the literature are reviewed, and 2 additional cases are presented.

Careful consideration of the reported cases suggests that awareness of the syndrome leads to more decisive action, with a consequent decrease in mortality.

Since splenic rupture occurs in infectious mononucleosis after minimal trauma, repeated abdominal palpation and strenuous exertion while the spleen is enlarged are contraindicated.

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MEDICAL PROGRESS

PHYSIOLOGY (Concluded)*

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SPECIAL CIRCULATIONS

The Liver

The unusual nature of the hepatic circulation has been emphasized by recently acquired evidence[108] that the oxygen supply to the liver may constitute the dominant factor determining whether shock becomes irreversible. It has been stated that the liver depends in large measure on portal blood for its oxygen,[104] and that increasing the oxygen content of portal blood by transfusion of arterial blood into the portal circulation protects the recipient against death from shock.[105] This view is in keeping with the classic anatomic opinion that the portal circulation is the major source of blood for the liver sinusoids. There can be no doubt, however, on anatomic grounds alone, that the hepatic artery also supplies the sinusoids, through anastomoses via the bile capillaries, direct arterioportal venous anastomoses and intralobular anastomoses.[106–109] Pathologically, the variable results of occlusion of portal or arterial vessels suggest that the relative contribution of the two vessels indeed varies. Grindlay, Herrick and Mann[110] measured portal inflow and hepatic venous outflow by the thermostromuhr method and found total flows averaging 100 cc. per 100 gm. of liver per minute, the hepatic artery flows constituting from 10 to 30 per cent of the total flow in the nonanesthetized animal. In anesthetized animals it was significantly higher, at times even exceeding the contribution of the portal vein.

This concept of dynamic variability of the portal circulation has much other evidence to support it. Rein and his co-workers[111] obtained flows of 62 to 75 cc. per 100 gm. per minute by the thermostromuhr method, with much the same partition between arterial and portal venous contributions. They also noted that, when adrenalin was given, more blood left the liver than entered it, so that the liver discharged as much as 60 per cent of its weight in blood. Bauer et al.[112] confirmed this finding in perfused livers and concluded that in the dog liver outflow is controlled by a variable sluice mechanism at the hepatic venous side of the circulation that could be closed by histamine or opened by epinephrine or sympathetic-nerve stimulation. In much the same kind of study McMichael[113] found that a portal venous hypertension followed administration of epinephrine, presumably by vasoconstriction in the ramifications of the portal system in the liver. Wakim[109] saw on microscopical examination of the liver in situ an intermittence in the activity of lobular sinusoids, which could remain relaxed and engorged with stagnant blood only to expel it later. All the foregoing evidence points strongly to a storage function in the operation of liver circulation.

Employing an ingenious adaptation of the Fick principle combined with catheterization of the hepatic vein in man, Bradley[114–116] has estimated the minute volume of the hepatic circulation. Assuming that the dye bromsulfalein is removed from the blood stream by the liver and no other organ, one can calculate liver blood flow by dividing the total quantity of the dye removed from the circulation in a given time by the difference in the concentration of the dye in systemic and hepatic blood. The total amount removed Bradley determined by the quantity of the dye injected in a given time when the rate of injection was balanced against the rate of removal in the liver to give a constant concentration in the blood. Values so calculated ranged from 1085 to 1845 cc. per minute in the adult human subject, with an average figure close to 1500 cc. per minute. Considering the average human liver to weigh 1500 gm., flows of 100 cc. per 100 gm. per minute were thus found, just as in the dog. After sympathectomy for hypertension in man, liver flow as measured in this manner rises markedly provided blood pressure is maintained, indicating that in man as well as in animals the liver circulation is under sympathetic control.[116] Since cardiac output in man averages about 5.4 liters per minute in the basal state, it is apparent that hepatic circulation constitutes a significant proportion of the whole, equaling in fact the total renal blood flow. The two together make up very close to half the total cardiac output. Even if it is admitted that the liver is, by virtue of its portal supply, out of the main circulatory channel and thus unable to contribute greatly to the peripheral resistance by which systemic blood pressure is regulated and that its circulation must be subject to significant passive or secondary changes resulting from gastrointestinal circulatory regulation,[117] the liver circula-

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tion must be considered as one of the important regions where a special circulation significantly conditions general hemodynamics.

Comparing hepatic blood flows as estimated above in normal nonpregnant women with those in normal pregnant women at various stages of gestation, and with blood flows in pregnant women with toxemia, Munnell and Taylor[118] found that pregnancy was without influence on hepatic circulation. In 6 toxemic women one exceedingly high rate of 4070 cc. per millimeter was found — almost exactly twice the highest value found in the normal pregnant group. Whereas no more than suggestive of a change in liver circulation in toxemia of pregnancy, the finding deserves further elucidation.

Another method of assessment of liver circulation has been devised by Lipscomb and Crandall.[119] Urea excretion is measured by urine collection, and this quantity divided by the portal-hepatic venous difference in urea, assuming that urea is produced in the liver and quantitatively excreted by the kidneys. Despite certain inherent difficulties that magnify the error in analysis of urea concentration (the portal-hepatic urea difference is small, averaging close to 1.0 mg. per 100 cc.) the procedure was found to check reasonably well with the bromsulfalein method. Myers[120] estimated hepatic flow by the urea method in 10 subjects and obtained an average of 1.0 liter per square meter of body surface per minute. With the bromsulfalein method he produced an average flow of 0.8 liter per square millimeter per minute, comparing well with Bradley's value of 0.9 liter.

Other Special Circulations

Catheterization of the renal vein has permitted studies of the extraction of oxygen and sodium para-amino hippurate by the kidneys for validation of other methods[121] and makes possible the assessment of the influence of intra-abdominal and venous pressure upon kidney function.[122] Studying the renin content of blood obtained by catheterization from the renal vein of 12 hypertensive patients and in comparable samples from 12 normal subjects, Haynes, Dexter and Seibel[123] found small but detectable amounts of renin in 7 of the 12 in each group. Superficially considered at least, this finding does not support the hypothesis that renin is the hormone responsible for human hypertension.[123] The observation that the Cournand catheter sometimes passes into the coronary venous sinus, as shown by its radiologic position and by the low oxygen content found in blood samples obtained when it is in that position, has encouraged attempts to measure the coronary blood flow in man.[124, 125] These have been based on Kety and Schmidt's[126] method for measurement of cerebral blood flow. When low concentrations of nitrous oxide are rebreathed for a few minutes and serial samples of arterial blood and internal jugular blood are obtained, a rising curve of the concentration of nitrous oxide in the arterial blood flow is inscribed, leveling off as equilibrium is reached. The curve of internal jugular blood concentration reaches the same maximum but at a later time and along a less steep curve, because of the loss of nitrous oxide to the brain by simple solution therein. If the nitrous oxide tension at the point of equilibrium is known, the total amount of the gas dissolved in a given weight of brain can be calculated from previously determined solubility curves. The arteriovenous nitrous oxide difference can be calculated from the area of the difference between the two curves of arterial and venous concentrations or by means of acceptable simplifications. Again the basic Fick calculation gives the cerebral blood flow in terms of unit volume of brain tissue. Measurement in monkeys checks reasonably well with determination by direct flowmeters.[127] Once the blood flow has been determined, it becomes possible to reverse the usual Fick calculation, and from this value and from direct determinations of arterial and internal jugular blood oxygen levels, to calculate the oxygen consumption of the brain.[128]

The Lungs

In the calculation of pulmonary blood flow by Vandam, Bing and Gray[101] it is assumed that blood returns from the lungs almost fully saturated (95 per cent). At operation these authors have had several opportunities to obtain pulmonary venous blood samples that proved as fully saturated as was expected, and Dr. Arnold Johnson, at the Children's Memorial Hospital, Montreal, has passed a catheter through an interventricular septal defect into the left auricle and there obtained arterialized blood 95 per cent saturated with oxygen. By inserting the catheter far enough into a branch of the pulmonary artery to obstruct it, Dexter and his co-workers[94] withdrew blood that was 95 to 100 per cent saturated with oxygen. They drew the logical conclusion that pulmonary capillary blood had been drawn backward into the catheter.[94] This is not the opinion of Taussig and Blalock,[129] however, who conclude as follows:

> We do not mean to imply that the lack of circulation to the lungs is the sole cause for the production of cyanosis and polycythemia, or that in the presence of polycythemia all of the blood which passes through the lungs is in effective contact with the oxygen in the alveolae (*sic*). On the contrary, the change in the patient's color when he is anesthetized and is receiving high concentrations of oxygen, offers clear evidence that without any fundamental alteration in the course of the circulation the oxygen saturation of the arterial blood may be greatly increased. In an attempt to analyze these factors, samples of arterial blood have been obtained prior to operation but after the patient was anesthetized and was receiving oxygen in high concentration. These samples have shown a striking increase in the oxygen saturation due to an increase in the oxygen content of the arterial blood, but, as is to be expected, there was no change in the oxygen capacity. These samples are truly basal, a condition which it is virtually impossible to obtain during an arterial puncture made when a child is

conscious. These values are probably influenced both by inhalation of a high concentration of oxygen and by the basal condition associated with anesthesia, and possibly by the anesthetic agent. The relative importance of these factors is not easy to determine. Nevertheless, the fact that there is a rise in the oxygen saturation of the arterial blood when the patient is given a high concentration of oxygen is in accord with the existence of pulmonary factors emphasized by Lundsgaard and Van Slyke in their classical studies on cyanosis.

Such a difference of opinion emphasizes how limited knowledge of the precise details of the pulmonary circulation, especially in man, has been, and shows how the new technics will prove of value, both by raising problems and by providing their solution.

The technic of catheterization has been combined with pressure recording by means of the Hamilton or other manometers, to make possible, for the first time in man, a direct quantitative study of the pressure relations of the pulmonary circulation.[130] In 77 catheterizations in 70 subjects, mean right arterial pressures varying between −2 to + 2 mm. of mercury were found in recumbent normal subjects. Pulse pressures ranged from 4 to 8 mm. of mercury. Curves showed peaks of pressure associated with auricular systole, closure of the auriculoventricular valves and accumulation of blood in the auricle during ventricular systole. The second peak is followed by an abrupt fall in pressure with descent of the ventricular base; after the third peak of pressure there is a rapid fall as interventricular pressure falls, and the phase of early diastolic filling of the ventricle begins. In the right ventricle maximum systolic pressures in normal subjects range between 18 and 30 mm. of mercury, averaging 25 mm. Among patients with lung disease, increased systolic pressures were found in the right ventricle in 14 out of 19 patients with chronic pulmonary fibrosis in the absence of failure of the right side of the heart and without elevation of diastolic pressures in the right auricle or ventricle. Patients after pneumonectomy or pneumothorax had normal pressures. A marked elevation in right ventricular pressure was found in a single patient with mitral-valve disease without evidence of right ventricular failure.

In the presence, however, of venous congestion coincident with right ventricular failure, diastolic pressures in the auricle and ventricle rose (7 to 9 mm. of mercury in 1 case), diminishing markedly the normal gradient of pressure between the peripheral veins and the right auricle. The intraventricular pressure curves showed characteristic alterations in contour, one of the most constant being an accentuation of the early diastolic "dip" in the ventricular and auricular pressure curves due to the sudden relaxation of the ventricle.

These findings have significance when considered from the standpoint of pulmonary cardiodynamics: since the outputs of the right and left ventricles are equal over long periods, the pulmonary circulation in general equals the systemic, which means in turn that pulmonary resistance must be very low indeed to permit such a large flow at such low pulse pressure; whereas under most circumstances there is equal output from the two ventricles, the capacity and flexibility of the small vessels of the lungs are such that considerable variations in the quantity of blood in the lungs can occur, so that during accumulation or discharge of this stored blood, right ventricular output may temporarily exceed or be less than left ventricular output. Quite the reverse, alterations of right or left ventricular output can influence the quantity of blood in circuit through the lungs.[131]

Whether the lung vessels have a clear-cut vasomotor regulation was still left in doubt by these studies, but Cournand et al.[132] have recently found evidence of another type of some kind of active regulation of peripheral resistance in the lungs. When normal adult males breathed mixtures of 10 per cent oxygen in nitrogen instead of room air, mean pulmonary artery pressure rose from 13 to 23 mm. of mercury, diastolic from 6 to 13, and systolic from 22 to 35. Calculated pulmonary resistance doubled. The maximum increase in pressure was reached within two to four minutes; the elevation of pressure remained as long as the subject breathed the low oxygen mixture, and fell rapidly back to normal as room air was readmitted to the subject. Cardiac output fell as much as 20 per cent. Animal experiments by other workers suggest that the phenomenon is an expression of a direct sensitivity of the pulmonary arterioles to oxygen lack, which serves to direct blood from nonaerated portions into regions of better oxygenation, where arterioles are in consequence relaxed.

Renal Disease in the Light of Recent Studies on the Renal Circulation

With the report by Bywaters and Beall,[133] in 1941, of 4 cases of azotemia, oliguria and finally fatal anuria following crushing injury to the extremities sustained in the bombing of London, numerous workers began to investigate the clinical and pathological features of many different conditions that give rise to renal shutdown. Bywaters and Dible[134] described the typical pathological features of crush syndrome in which there is pallor of the renal cortex and degeneration of the convoluted tubules, which are often filled with desquamated debris and pigment-containing casts; the pigment was identified as myoglobin, a substance related to hemoglobin and presumably liberated by the crushing injury to large amounts of muscle. They speculated on the part played by the pigment casts in the production of oliguria by mechanical obstruction or by toxic effect on the kidney, and came to the conclusion that it was probably an aggravating factor but not necessarily the entire cause; the similarity of this lesion to that seen in

blackwater fever, in which there is intravascular hemolysis, was noted. They also drew attention to the fact that the crush syndrome was not a new condition as they had previously thought but had been recognized by German clinicians and pathologists in World War I. A review of these cases was published by Minami[135] in 1923. De Navasquez[136] reported 3 fatal cases of transfusion reaction and pointed out that 2 of these patients had had alkaline urine; experimentally, when hemoglobin was injected into rabbits, animals with acid urine actually excreted more iron in their urine than those with alkaline urine. He also found insufficient tubular blockage to account for the anuria. Hence he concluded that mere hemoglobinuria was not the sole cause of anuria and that hemoglobin was not precipitated in undue amounts owing to the acidity of the urine; he suggested that diminished glomerular circulation might be the cause of the anuria. In 1942 Yuile[137] reviewed the literature on hemoglobinuria and concluded that hemoglobin was able to pass into the glomerular filtrate only after damage to the glomeruli, possibly because of anoxia. He also noted that not all cases of "transfusion kidney" at autopsy had pigment casts. Foy and his associates[138] suggested that the anuria in all hemoglobinurias, sulfonamide reactions, favism, crush injuries and so forth fell into a common category, pointing out the rather similar pathological findings and suggesting that the lesion was caused by dehydration with diminished blood volume and renal circulation; in their opinion the pigment casts were only an aggravating factor.

Meanwhile in 1942 Barnes and Trueta[139] had been investigating the effects on the circulation of the rabbit's hind leg produced by a rubber-covered wire tourniquet applied to the thigh for four and a half hours. They found that this produced a severe spasm of the vessels of the affected limb for periods up to seventy-two hours (well beyond the period of shock and hemoconcentration), as well as a spasm of the arteries of the opposite limb. This was demonstrated by arteriography, thorotrast being injected into the abdominal aorta. During the laparotomy to expose the aorta, it was noted that the kidneys were pale and blanched on their surfaces but the authors were unable to pursue this observation at that time. Trueta et al.[140, 141] returned to this investigation in 1945. The subjects of the experiment were rabbits, and a variety of experiments were carried out. Serial radiography using thorotrast as the radiopaque material, revealed, after release of the tourniquet on the left hind limb in the intact animal, that the calibers of the renal artery and the femoral artery of the left side were greatly reduced, whereas there was some dilatation of the superior mesenteric artery. At laparotomy they noticed that the renal cortex was pallid, although the renal vein was redder than normal, often containing bright-red central streamlines and showing pulsations almost like an artery, suggesting a rapid circulation through part of the kidney at the expense of the cortex. This was confirmed by study of cineradiographs, which showed that the circuit time of the radiopaque mass through the affected kidney might be half that of the normal and the size of the renal artery was reduced to a quarter its normal size. The cortex of the affected kidney was less opaque than the medulla during the phase of the renal circulation when one would expect the cortex to be more opaque; furthermore, in the left kidney at that time it was noticed that although the thorotrast appeared in the renal vein sooner than normal, it took a longer time for the renal artery to be completely emptied. These observations suggested some sort of circulatory by-pass through the medulla of the affected kidney. To study this more fully dyes and particulate matter were injected into the arterial side of the circulation during the period of cortical ischemia; these substances stained and filled the medulla completely and yet did not appear in the cortex or stain the surface as in the normal kidney. Histologic examination of the kidneys after injection of the India ink localized the pigment in the vasa recta of the medulla and in subcortical vessels of considerable size. These changes in the renal circulation could also be evoked by the intravenous injection of adrenalin, pitressin or staphylococcus toxin; with the injection of the last substance an extreme degree of cortical ischemia was produced, resulting in a pathologic lesion identical with that found in bilateral cortical necrosis. Similar but less extensive cortical ischemia was found in animals that had been bled sufficiently to produce shock. Stimulation of the central end of the divided left sciatic nerve or the distal end of the divided splanchnic nerves also caused renal cortical pallor and medullary circulatory by-pass. When, however, the splanchnic nerves were previously divided the renal circulation in the tourniquet of otherwise stimulated animals remained relatively unchanged, although the femoral artery of the affected limb and the mesenteric vessels showed some constriction.

From these experiments it was concluded that a similar mechanism producing cortical ischemia could be the cause of the azotemia and oliguria seen in renal injury due to sulfonamides and transfusion of incompatible blood, Weil's disease, cholera, crush syndrome, hysterical anuria and so forth. In support of this theory a considerable body of experimental, clinical and pathological evidence has been presented in recent years. In 1944 Richards[142] found in studies of the circulation in traumatic shock in man that when cardiac output dropped to about half normal renal blood flow fell to a tenth or twentieth of normal, and simultaneously that the urine output fell; he saw renal failure only once in these cases, however. Selkurt[143] cannulated the renal vein via the jugular to avoid local trauma and

spasm, and then made simultaneous recordings of renal clearance and blood flow during shock produced by hemorrhage. He found that when the pressure had fallen to 40 mm. of mercury the renal circulation had dropped to 11 per cent of normal, but clearances of para-aminohippuric acid and creatinine were zero. However, when the blood pressure was raised by transfusion to about 80 mm. of mercury and the renal blood flow was 80 per cent of normal, the clearance of para-aminohippuric acid and creatinine was not restored to normal but still remained nearly at zero. Selkurt could not explain this to his satisfaction, but it now seems that it was due to the re-routing of the renal circulation as described by Trueta et al.[141] Pathologically the kidneys of these dogs showed cloudy swelling and early degenerative changes in the tubular cells. Scarff and Keele[144] found in 1943 that clamping the renal artery in rabbits for an hour after the other kidney had been removed resulted in azotemia and oliguria, frequently going on to fatal uremia, and produced degeneration of the tubules, not unlike that found in crush syndrome and so forth, although it principally affected the proximal convoluted tubules rather than the lower part of the nephron. Corcoran and Page,[145] reviewing their experimental work on dogs in relation to post-traumatic anuria in 1936, reported that injection of hemoglobin or myoglobin into dogs and rats caused variable damage, which was frequently not serious even when the urine was acid; however, if this were accompanied by shock to produce renal ischemia, most of the animals died of anuria. From this they concluded that renal ischemia was the important factor in the pathogenesis of this condition. Lauson, Bradley and Cournand[146] found by studies of inulin and diodrast clearance in shocked men that there was a fall of renal blood flow out of all proportion to the fall of systemic blood pressure, which, they calculated from estimated renal artery and vein blood pressure and renal blood flow, was due to increased resistance in the kidney. Darmady et al.[147] and Darmady[148] analyzed a total of 17 cases of traumatic anuria occurring in the British forces in France in 1944–1945. All showed a clinical picture, developing about five or six days after injury, distinguished by azotemia, oliguria, elevated serum potassium and occasionally mild hypertension. Most of these patients had received blood transfusions at some time, but no reactions were recorded. In the 12 fatal cases the authors found the typical pathological features of degenerated convoluted tubules and frequent pigment casts in the tubules, whereas the cortices were pale and glistening and the glomeruli rather avascular. They suggested that the lesion was due to renal anoxia, and noted that in 2 patients who recovered there was dramatic improvement after splanchnic block.

Maegraith, Havard and Parsons[149] came to similar conclusions in 1945, after comparing the pathological features of blackwater fever, incompatible transfusion, bilateral cortical necrosis, Weil's disease, hepatorenal syndrome, renal injury due to sulfonamides, crush injury and cholera. Duff and More[150] analyzed 71 cases of bilateral cortical necrosis of the kidney that came to autopsy, ascribing the pathogenesis to renal ischemia of unknown origin, which is possibly an individual idiosyncrasy to some stimulus or toxin. Mallory,[151] in 1947, and Lucké,[152] in 1946, after extensive analyses of over 500 cases of renal shutdown occurring in the American forces during the war, ascribed the renal lesion primarily to anoxia, although Mallory believed that the factor of mechanical blockage of the tubules could not be completely excluded and was certainly an aggravating factor.

Trueta et al.[141] ascribed the oliguria to reduced glomerular circulation and consequent low filtrate formation, and also to increased reabsorption in an unselective fashion from the tubules due to the increased blood flow in the vasa recta and the fact that the tubules are damaged. In support of the latter theory they cited the work of Dunn and Polson[153] in 1926 and Richards[154] in 1929, who found that in tubules damaged by uric acid or bichloride of mercury there is increased reabsorption of water with oliguria and with loss of normal impermeability to phenolsulfonephthalein. By this process Lucké[152] accounted for the presence of tubular casts, which he considered a result rather than a cause of oliguria, owing to excessive reabsorption of water. Brun, Knudsen and Raaschou[155] studied the effect of syncope on renal clearance of inulin and diodrast in man and noted that although renal plasma flow returned to normal within a few minutes, there was oliguria lasting for about sixty to ninety minutes. They concluded that this was due to increased tubular reabsorption and suggested that it was induced by the antidiuretic hormone of the posterior portion of the pituitary body, for blood withdrawn from these men during the period of oliguria produced oliguria in normal controls. There was also increased chloride excretion similar to that produced by the antidiuretic hormone, and in 2 men with diabetes insipidus the postsyncopal oliguria was less marked than normal. Trueta and his co-workers[141] were undecided whether the cause of the ischemia was toxic or neurogenic, or both. Page[156] found a vasoconstrictor substance in blood during shock produced in dogs by trauma, hemorrhage or burns that caused vasoconstriction in the perfused vessels of a rabbit's ear; he decided that it was not histamine or angiotonin because when the vessels of the perfused ear were fatigued by repeated injections of this substance they still constricted with histamine or hypertensive plasma, and the converse was also found. This substance could be obtained in the blood after removal of the kidneys, adrenal glands and spinal cord. Eggleston[157] found a substance in muscles of dog's legs

rendered ischemic by some hours of binding with a tourniquet, which depressed creatinine clearance in the kidney; it was also observed in muscles of dead animals between four and ten hours post mortem, after which time it disappeared. This substance was unstable and could not be dialyzed. If injected slowly through the portal vein it produced no effect suggesting that it was detoxified by the liver. Lucké[162] postulated that this explained the occurrence of hepatorenal syndrome after operation on the biliary tract when there is presumed to be some alteration of liver function. That nervous inhibition of urine function can occur is shown by the experiments of O'Connor and Verney[158, 159] in 1942 in which they demonstrated two types of emotional inhibition of water diuresis in dogs produced by sounding a horn or faradic stimulation of the skin, one being rapid and the other slow and delayed. The rapid type is abolished by sympathectomy, and the authors suggest it is due to renal ischemia produced by impulses traveling in the splanchnic nerves; the second is due to the antidiuretic hormone of the pituitary body. Similarly, Charcot[160] reported cases of hysterical anuria in 1877. On the other hand Smith,[161] using clearance studies of renal circulation, showed that increased sympathetic tone induced by elevating a previously supine man on a tilt table caused renal vasoconstriction primarily in the afferent arterioles; a similar effect was produced by psychomotor stimuli.

Having found that such a medullary by-pass existed Trueta and his co-workers[141] investigated the anatomy of the renal circulation by means of neoprene casts, some being made in rabbit's kidneys and others in those of human subjects. To show the side of this by-pass they injected the renal arteries of rabbits after producing cortical ischemia experimentally. At times this still did not enable them to demonstrate fully the flow into the vasa recta of the medulla, so that they first injected large quantities of thorotrast into the rabbit's arterial system, which they found completely blocked the cortical glomeruli. Using these technics in the rabbit's kidneys and by straight injection technics in human kidneys they found that the by-pass in circulation occurred through what Heggie[162, 163] called the juxtamedullary glomeruli. In these glomeruli the circulation of the nephron is somewhat different to that which occurs in the cortical glomeruli. In the latter, as originally described by Bowman[164] in 1842, the blood travels through the interlobular arteries and thence to the afferent arterioles and the capillaries of the glomerulus from which emerges the efferent arteriole, which then breaks up into the capillaries surrounding the convoluted tubules, and after following the descending and ascending limbs they reunite to join the venous side of the circulation. In the juxtamedullary glomeruli the following differences were noted. First of all it was found that the efferent arteriole—unlike that in the cortical glomeruli—was larger than the afferent arteriole. Secondly, the efferent arteriole does not break up into the usual capillaries that surround the convoluted tubules, but immediately forms large vessels which descend into the medulla as the vasa recta. These vessels, the vasa recta, were always poorly described anatomically before this, since they were difficult to inject. Trueta et al.[141] found that after descending for varying distances into the medulla they hook back upon themselves, forming curious "H" and "Y" forms, and that when they reach the juxtamedullary region they reunite to join the venous system. These vessels form very extensive thin-walled channels about the ascending and descending limbs of the tubules descending into the renal medulla. In this connection it was pointed out by Trueta et al.[141] that Peter[165] showed in 1909 that the tubules of the cortical region only descended into the outer fringes of the medullary zone of the kidney, whereas those from the juxtamedullary-zone glomeruli were much larger, descending deeply into the medulla, and had a much longer loop of Henle. Hence these tubules are surrounded by the vasa recta through which the entire circulation of the kidney passes during the periods of cortical ischemia. Thus, there is further support for the theory that greater tubular reabsorption is a partial cause of traumatic anuria.

In their anatomic studies of the renal circulation in man Trueta et al.[141] noted in many cases in these juxtamedullary glomeruli that there was marked degeneration of the glomerular capillaries, which at times was so extreme that there remained only a direct connecting channel, with a muscular coat, between the afferent and the efferent arteriole, the site of the degenerated glomerulus being marked only by a slight curve in the vessel. The vessels were only found in kidneys in which there was some degenerative process, and had been previously reported by MacCallum[166] in 1939. In all probability these vessels were what caused all the dispute between the earlier anatomists regarding whether or not Ludwig's arteriole, or the arteriae verae rectae, existed. They found no evidence of any other types of nonglomerular circulation.

Oliver[167] observed these degenerated glomeruli with a direct continuity between the afferent and efferent arteriole to be especially prevalent in cases of chronic Bright's disease. From this fact, and considering the role of renal ischemia in the experimental production of hypertension as shown by Goldblatt[168] and numerous subsequent workers, Trueta et al.[141] suggest that there is some connection between this by-pass of normal renal circulation and the etiology of essential hypertension. They postulate that in normal persons, owing to varying stimuli, there is frequent re-routing of the renal circulation through the juxtamedullary glomeruli. When this mechanism is called into being too

frequently it may ultimately lead to glomerular degeneration, a constant by-pass of the cortical glomeruli with resulting ischemia and hypertension. Whereas the studies of Smith and his collaborators have made it difficult to maintain any theory of essential hypertension based on gross glomerular ischemia of any persistence, the possibility, of course, remains that circulation through specialized vessels not included or lost sight of in the usual clearance methods of measuring renal blood flow is the crucial factor in the origin of essential hypertension.

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MASSACHUSETTS MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND SIXTY-SEVENTH ANNIVERSARY

May 25, 26 and 27, 1948

THE one hundred and sixty-seventh anniversary of the Massachusetts Medical Society was observed at the Hotel Statler in Boston on May 25, 26 and 27, 1948.

Seventeen hundred and forty-one physicians, 219 ladies and 495 exhibitors were registered.

The Committee on Membership met in Parlor C at 4:30 p.m. on the afternoon of May 24. The Supervising Censors met in Parlor C at 5:00 p.m. This meeting was followed by the Cotting Supper in Parlors A and B, which was attended by 236 councilors. The annual meeting of the Council was held in the Georgian Room at 7:00 p.m. with 235 councilors in attendance, as recorded in the attendance books.

Tuesday, May 25

The first general session opened at 9:00 a.m. in the Georgian Room under the co-chairmanship of Dr. Dwight O'Hara and Dr. Frederick S. Hopkins.

The one hundred and sixty-seventh annual meeting of the Society was held in the Georgian Room at 11:00 a.m., President Edward P. Bagg presiding. The attendance was about 400. Dr. Bagg spoke on "The State of the Society," after which the annual oration, "The Responsibility of Medicine in the Propagation of Poor Protoplasm," was delivered by Dr. Allen S. Johnson. (This lecture appeared in the May 27 issue of the *Journal.*) The annual luncheon was served in Parlors A, B and C to 104 fellows.

The second general session was held in the Georgian Room at 2:00 p.m. under the co-chairmanship of Dr. W. Richard Ohler and Dr. Lewis M. Hurxthal. The Shattuck Lecture was delivered at 8:00 p.m. by Dr. C. Stuart Welch, of Boston, on the subject "Surgery in the Aged." (This lecture appeared in the June 10, 1948, issue of the *Journal.*)

Wednesday, May 26

The third general session was held at 9:00 a.m. under the co-chairmanship of Dr. Charles J. Kickham and Dr. Roy J. Heffernan.

At noon certain of the sections held their luncheons, followed by their annual meetings. The Section of Medicine, under the chairmanship of Dr. Francis C. Hall, met in Parlor A. The attendance was 142. The Section of Pediatrics, under the chairmanship of Dr. Lewis W. Hill, met in Parlor B, with 60 members in attendance at the luncheon and 75 at the scientific meeting following. The Section of Radiology, under the chairmanship of Dr. Hugh F. Hare, met in Parlor C, with 52 in attendance at the luncheon and 75 at the meeting. The Section of Physiotherapy met in the Hancock Room under the chairmanship of Dr. William D. McFee, with 15 members present.

At 2:00 p.m. the fourth general session was held in the Georgian Room, under the co-chairmanship of Dr. William B. Castle and Dr. Stanley J. G. Nowak.

The annual dinner was held in the Georgian Room at 7:00 p.m. with 456 in attendance. The speaker of the evening was Mary Ellen Chase, Professor of English Language and Literature at Smith College, who delivered an address entitled "The Country Doctor on the Maine Coast."

Thursday, May 27

The fifth general session was held in the Georgian Room at 9:00 a.m. under the joint chairmanship of Dr. John J. Poutas and Dr. Claude E. Welch.

At noon the remaining scientific sections held their luncheons and meetings. The Section of Surgery, under the chairmanship of Dr. George S. Reynolds, met in the Salle Moderne, with 140 in attendance. The Section of Obstetrics and Gynecology met in Parlor A under the chairmanship of Dr. Arthur T. Hertig, with 50 in attendance. The Section of Dermatology and Syphilology met in Parlor C under the chairmanship of Dr. Austin W. Cheever, with 38 in attendance. The Section of Anesthesiology, under the chairmanship of Dr. Urban H. Eversole, met in Parlor B with 53 members attending.

The sixth general session was held at 2:00 p.m. under the joint chairmanship of Dr. John Fallon and Dr. George S. Reynolds.

An exhibition of works of art by members of the Massachusetts Physicians' Art Association was on view throughout the meeting.

The special list of officers, standing and special committees, councilors, censors, admissions and deaths is appended.

H. Quimby Gallupe, *Secretary*

Annual Meeting of the Society

The one hundred and sixty-seventh annual meeting of the Massachusetts Medical Society was called to order by the president, Dr. Edward P. Bagg, in the Georgian Room of the Hotel Statler, Boston, at 11:00 a.m., on May 25, 1948. Approximately 400 fellows were present.

The Secretary submitted the record of the 1947 annual meeting, held on May 20, 1947, and pub-

lished in the August 28, 1947, issue of the *New England Journal of Medicine*, and moved its acceptance. The motion was seconded and so voted.

President Bagg brought up the matter of the amendments to the by-laws and stated that in the printed form mailed to the fellows two mistakes had been noted at the Council meeting on the previous day — namely, the use of "Inc." following the two appearances of "The Massachusetts Medical Service" — and that these should be omitted. He also noted the omission of the Vice-President in the list of officers.

Dr. Bagg then read Article II of the Articles of Incorporation of the Society:

> The Fellows of said Society shall have full power and authority to make and enact such rules and by-laws for the better government of the said Society as are not repugnant to the laws of this Commonwealth, and to annex reasonable fines and penalties, etc. The Fellows at their Annual Meeting, whenever such a number shall be present as the regulations of the Society may require [being 100, and there are 100 present] may revise, alter, enlarge, and repeal the by-laws of the said Society as the major part of the Fellows present may see fit, and not otherwise.

He then urged the adoption of these amendments (Appendix No. 1), that the work of the Society might be carried on more successfully.

The Secretary moved that the amendments to the by-laws, with the corrections already noted and made, be adopted. The motion was seconded.

Dr. Carl Bearse, Norfolk, then noted two more omissions as follows: Chapter VI, Section 4 under duties of the Secretary, "he shall perform such other duties as the Society or the Council may require," and again under Chapter VI, Section 7, under duties of the Executive Secretary, "he shall hold office at the pleasure of the Executive Committee."

Stressing the point that a committee should screen by-law changes and that the proposed changes were not urgent, Dr. Bearse moved that they be tabled until the next annual meeting. The motion was seconded. Dr. Bagg said that the motion was not debatable and asked for a show of hands. The Secretary counted and reported the motion carried.

Dr. Bagg registered his disapproval of the action and said he wished that the by-law changes could have been adopted.

Dr. Elmer S. Bagnall, Essex North, said that he believed that the fellows did not realize, when they voted, what the conditions would be and moved reconsideration. The motion was seconded and on a show of hands the motion was carried.

Dr. Albert A. Hornor, Suffolk, then asked for a reading of the motion to be re-considered. (The motion was read.)

Dr. Dwight O'Hara, Middlesex South, moved the adoption of the by-laws as amended. The motion was seconded.

Dr. Bagg then asked for the inclusion of the omissions as related by Dr. Bearse above. It was moved and seconded that these inclusions be made. It was so voted.

The Secretary reported on the membership of the Society as follows:

> In May, 1947, the membership of this Society was 6256. There was a gain of 403 new members, and 9 reinstatements, making a total of 6668. Our losses consisted of 88 deaths, 35 resignations and 7 deprivations. The total membership in the Society, as of May 25, 1948, is 6538.

The Secretary then commented upon the *Directory* of the Society as follows:

> The directory now in use was published last year and is correct as of July, 1947. It has in it all the information concerning the membership of the Society that the Society deems essential, and is issued under the direction of the Committee on Publications. The directory is available to members of the Society on request, and extra copies may be obtained by purchase from the office of the Society.

At the request of the President, the vice-president, Dr. Charles J. Kickham, Norfolk, assumed the chair and Dr. Bagg gave the following address:

> The twelve months just completed undoubtedly will be judged in historical perspective as a period of transition for the Massachusetts Medical Society. In the loss just previously of our exceptionally able secretary, Michael A. Tighe, a severe handicap was imposed upon the efficient conduct of the Society's business, because he carried so many of the important details in his head. Coincidentally, the office assistant on whom he in turn had leaned was compelled by ill health to leave our employ.
>
> However, President Dwight O'Hara, as his parting contribution, furnished the Society and the incoming regime with a capable new Secretary, Joseph Garland. The latter was courageous enough to accept the challenge to do what he could to put our affairs in order. You yourselves know how well he succeeded at no small sacrifice to himself. But I can assure you, from the point of view of harassed presidential inexperience, that his willingness and patience were exceeded only by his good nature and all-around reliability. Somehow we survived that first council meeting, though the margin was slim.
>
> And then in September a second severe blow again deprived the Society of a highly esteemed fellow, the able editor of the *Journal*, Robert N. Nye. Once more the secretaryship fell vacant when the Committee on Publications elected Joseph Garland to succeed him as editor-in-chief. The latter long had been interested and associated in the editing of the *Journal* and naturally chose to devote his full time to publishing rather than recording the minutes and managing presidents for the Society. There really was no question in his mind or in mine about how the interests of the Massachusetts Medical Society would best be served, so that genial Joseph was released unconditionally to reactivate the blue pencil.
>
> In behalf of the entire membership, hearty thanks are hereby extended to our new editor and erstwhile secretary for his prompt and loyal endeavors to help us in whatever role that was assigned to him.
>
> When the availability of H. Quimby Gallupe became known, his fine record in rectifying the disordered affairs of the Board of Registration in Medicine as secretary, and his wide acquaintance with medical practitioners of Massachusetts both in and out of the Society gained in this way, prompted his appointment early in December as secretary *pro tempore*. His nomination last month for the position of full-time secretary bears witness to the satisfactory character of his temporary ministrations. Under his supervision, the routine business of the Society now is handled with a minimum of friction by a superior

team of collaborators. Letters and queries are answered promptly, and there has been excellent co-operation with the other officers of the Society.

One of the epochal features of the transition stems from the change in by-laws that places the secretaryship on a full-time basis and in the scribe's hands responsibility for the work of his associates, the Director of Medical Information and Education and the Executive Secretary who pulls more than his own weight as office and business manager for the Society. In addition, Miss Gaston's Information Bureau, which has served such a useful purpose, has come under the Secretary's wing. Hereafter, all committee meetings will be cleared through him so as to prevent duplication in dating and accommodations.

In this manner, a continuity of policies and a proper regard for essential details not only will be attained but also will no longer depend upon officials who change from year to year. The presidency from now on should be a less demanding office, particularly when that officer is domiciled out of Boston. Both the Society and the incumbents thereby will gain. The Secretary will sit with practically all the Society's many committees to give advice when asked and to keep track of the proceedings. However, he will have the right to vote at only a few of them so that his power cannot become too great and thereby reduce the Society to just another example of eastern democracy such as Tito vouchsafes to the Serbs, the Croats and the Slovenes.

On July 1, 1947, the staid old Massachusetts Medical Society definitely broke with the past for the sake of improving public relations. The swift advance and dissemination of new facts in science during the twentieth century had effected a partial eclipse in the prestige of the medical practitioner. He himself was so busy meeting the demands of ailing fellow citizens and attempting to keep abreast of discoveries that cults and professional social workers were usurping his prerogative of preserving the health of the nation. Press relations were poor and legislators often were not much impressed with the doctor or what they conceived to be his motives. Smaller groups with more efficient organization were getting the attention and the votes.

In less than one year, I am glad to report, thanks to our energetic Director of Medical Information and Education, the situation, if not completely reversed, is greatly improved. The press now has ready access to facts and can check news items with authority before publication, and columns are open to us with unaccustomed liberality. The ink was scarcely dry on John Conlin's public-health diploma before he was in full swing in his new office. In less than six months he so hedged himself in with files, clippings and telephone calls than an expert stenographer, a press assistant and a sound scriber that could function at all hours were rushed to the rescue. His inspired management of the "pound law" campaign need not be re-told here, but the public as well as the legislators have once more come to understand that the medical profession, as a whole, is not venal or self-centered, and that the Massachusetts Medical Society's chief purpose is to further medical education and so to improve the standards of practice and to further the prevention of disease for one and all. Proud as I am of John's beginning, I have reminded him that the flesh has limitations and asked him hereafter not to spread himself too thin for efficiency in attacking important issues.

It is a pleasure to acknowledge the large debt of gratitude that we all owe to the ever-willing, always agreeable and yet astute Bob Boyd, who reserves the accommodations, satisfies hotel managers or hires perfect office girls all in his day's work. He has dealt so fairly with exhibitors that our display space is always oversubscribed by representatives who declare that they will come to Boston by preference so long as they can do business with our Robert. The balance sheet of the New England Postgraduate Assembly always shows black figures because we lend our business manager. More power to him, say I, and sometime spon more pay!

Those of you who have had occasion to sample the work of our stenographers know how well the officers and the interests of the Society have been served by the Misses Biggy, Kelly and Adler. They have outdone themselves in speed and accuracy during this transition period with a most commendable spirit of friendly co-operation in spite of many new faces, new problems and new equipment. With their help also, the excellent 1947 *Directory* was compiled in the midst of the turmoil.

Such, then, is the state of the Society at the present writing. The sedate mid-Victorian styling has been replaced with the "New Look" of the mid-twentieth Century. Heaven forefend that coming modes should drive her again to straight lacing and waspishness. Naturally, it costs more today to keep our Alma Medica as she should be kept. Our dues, as you know full well, have more than doubled. To what ends, you may ask with justice, and with what results?

Of course, quite a number of resignations were prompted by the receipt of this year's bills. But most of these fellows already had reached retirement age and most of the others were residents outside Massachusetts. It is a sad commentary on the assiduity with which the *New England Journal of Medicine* is read to report that the Treasurer received not a few irate queries regarding his authority, his intentions and why in blazes he should need more pay. In spite of all this, our membership continues to grow, having surpassed 6500, and more members had paid their assessments before the March 1 deadline than did so in 1947. Thanks to this splendid record, more than $22,000 has been turned over to the Boston Medical Library to assist in making more accessible one of the world's choicest collections of medical information. The new directors representing the Massachusetts Medical Society are already aiding in allocating this much-needed fund to best advantage.

A special committee is looking into the question of how we may assist the widows and dependents of fellows who have come upon hard times, as was originally recommended in partial justification of higher dues. Furthermore, in accordance with instructions, twice as much money was returned to the district societies this year.

Advances in pay for the employees in our office, together with retirement policies, have been put into effect according to previous authorization. The result was a considerable increase in our overhead expenses.

To cap the climax, the Commonwealth of Massachusetts now insists that we must pay the employment security tax on our own employees and those of the *Journal* retroactive to the middle of 1944, so that the financial status of the Massachusetts Medical Society may not prove to be so roseate as anticipated with the January 1 increase. The Treasurer may still find himself "standing in the need of prayer," if not of monies.

The reports of the numerous committees speak for themselves, but I should like to reiterate my admiration for the committeemen whose patience, ability and ready co-operation have opened my eyes during this enlightening year. Friendships formed in sharing assignments have more than compensated for the inevitable sacrifices and for geographic and meteorologic aggravations that impeded our efforts.

It is regrettable that the solution of the special services' problem was thwarted by economic considerations and the widespread lack of up-to-date hospital cost accounting. At least the definitions have been made clear and are widely distributed. Fellows who serve on individual hospital staffs must implement them locally wherever possible.

Judging from latest reports, there is small likelihood of the passage of compulsory health legislation in the current session of Congress, but this too is a problem that must continue under constant scrutiny.

Perhaps the most urgent item of unfinished business is that of our new headquarters, made more so by the need of the Medical Library for larger space. All year long Chairman Frank Ober has verged on but actually never achieved the gladsome report of a real-estate transfer that would provide us with a more suitable meeting place and rooms in which committees may function. If we do not acquire better accommodations soon, the proportion of district representation must be reduced. Otherwise, councilors will not have room to masticate the Cotting lunches, let alone hold rational converse with one another.

To these few admonitions, I join the best of wishes for the success of the incoming regime. The new officers

will find, as their predecessors found, that new problems will arise soon enough and frequently. I have full confidence that we are well set to meet whatever contingencies may arise.

Hippocrates, our father in medicine, composed the following precept (Chapter XII): "If for the sake of a crowded audience, you do wish to hold a lecture, your ambition is no laudable one, and at least avoid all citations from the poets. . . ." Accordingly, let me close this report in heartfelt prose: Fellows of the Massachusetts Medical Society, for the honor and privilege of serving you, I am deeply grateful.

Dr. Bagg resumed the chair and asked the Secretary to read the list of those fellows who joined the Society in 1898 and were still active, and requested those present to come forward and receive a gold pin as a memento to remember the occasion.

The Secretary read the following list of names:

Dr. George S. C. Badger, of Boston
Dr. Daniel C. Dennett, of Winchester
Dr. Clara P. Fitzgerald, of Worcester
Dr. Walter W. Fullerton, of Brockton
Dr. Joseph A. Hogan, of Lawrence
Dr. Myron L. King, of Cambridge
Dr. George C. Littlefield, of Webster
Dr. Alverne P. Lowell, of Fitchburg
Dr. Harry W. Luchsinger, of Great Barrington
Dr. George F. MacKay, of Dalton
Dr. William V. McDermott, of Salem
Dr. William D. McFee, of Haverhill
Dr. Harris P. Mosher, of Marblehead
Dr. Marion Nute, of Brookline
Dr. Joseph H. Pratt, of Boston
Dr. Jane D. K. Sabine, of Boston
Dr. Frederick W. Stetson, of Boston
Dr. Peter H. Thompson, of Boston.

The Secretary then offered the following motion:

According to Chapter I, Section 4, of the by-laws, two fellows of the Society, namely, Dr. Bagg and Dr. Gallupe, have nominated Sir Reginald Watson-Jones as an honorary fellow. This has been confirmed by the Committee on Membership and by the Council yesterday.

According to Chapter 1, Section 4, this confirmation should have been made at a previous stated meeting of the Council, but it was impossible to do so, owing to lack of time or sufficient foresight.

I move that, according to Article 2 of the Articles of Incorporation, this meeting alter the by-laws temporarily, that the granting of this fellowship may be made possible.

Dr. Bagnall seconded the motion, and it was so voted.

Dr. Gallupe moved that the Massachusetts Medical Society designate Sir Reginald Watson-Jones as honorary fellow. The motion was seconded.

Dr. Bagg spoke as follows:

I won't allow discussion of that. All those in favor, please say Aye. Those opposed, no. It is a vote. I have delegated Dr. Russell Sullivan as a page to bring the recipient to the dais.

Honored Fellow, if your knowledge of Latin is as rusty as ours, I am prepared to furnish you with a translation of what is inscribed on your diploma. I shall read it:

"Whereas it is usual for a Society devoted to the furtherance of medicine to elect to fellowship men of productive scholarship in this art, the Medical Society in the Commonwealth of Massachusetts, founded A.D. 1781, designates as Honorable Fellow Honorable Sir Reginald Watson-Jones, a man of recognized attainment in advancing the interests of science, and accords him all the privileges of the Society. The Society would express its desire that the recipient of this honor may communicate in the form of papers any original contributions made by him to the art of medicine. In token whereof the Council have ordered this Seal of the Society to be affixed, at Boston, Massachusetts, May 25, A.D., 1948, and in the one hundred and seventy-second year of the Independence of the United States of America."

I welcome you into fellowship.

Sir Reginald Watson-Jones replied as follows:

I am sorry that this election caused some little disturbance in the matter of the by-laws, but I am very glad that the by-laws situation was resolved so easily and smoothly. I can't tell you how deeply I appreciate the very great honor conferred upon me, the honor of being numbered among the very select few honorary fellows of this oldest medical society in the United States.

Whenever I make any contact with Boston, it seems to me that my friendships with the men of this city, the surgeons of this city and the doctors of this city, increase every time. My first contact was twenty-five years ago. I came here as an extremely unknown, unheard of man as a ship's surgeon on one of the Cunard ships, and I was received in the city as if I were a great leader of surgery. I was even taken out to a clambake. And every year since then, the number of my friends and the closeness of my friendship with those friends has increased.

This great honor that you have done me is one more mark of the warmth of friendship between surgeons on the two sides of the Atlantic. And I have only one hope, sir, and that is that in due course — I believe I can just make it — I may attend here to receive from the hands of the President of 1998, the medal of fifty years' membership in the Society.

The President then called for the delegates from the New England State Medical Societies. Dr. Robert A. Goodell, of Connecticut, responded as follows:

I do not know whether I am the only representative from the New England States, but I wish to have the honor and privilege of bringing you greetings from the Connecticut State Medical Society. Dr. James R. Miller, the retiring president, and Dr. Harvey, the president-elect, both wish me to extend greetings and best wishes, and I am sure that I am doing that also for the entire Society. I wish to congratulate you on your splendid program, and I trust if any of you have the opportunity to visit the meeting of the Connecticut State Medical Society, you will receive as cordial and as gracious a welcome as I have received here.

The President then introduced the incoming officers, after which the one hundred and fortieth annual oration, "The Responsibility of Medicine in the Propagation of Poor Protoplasm," was delivered by Dr. Allen S. Johnson, of Springfield.

Dr. Bagg then declared the one hundred and sixty-seventh annual meeting of the Massachusetts Medical Society adjourned at 12:30 p.m.

H. QUIMBY GALLUPE, *Secretary*

APPENDIX NO. 1

Amendment to By-Laws

To be approved by Annual Meeting of Council on May 24, 1948.

To be presented to the Massachusetts Medical Society at its Annual Meeting to be held on May 25, 1948.

Chapter IV, *Section 1*:

The Council shall consist of councilors chosen by the district societies, the president, vice-president, ex-presidents, president-elect, vice-presidents ex-officiis, secretary, treasurer, and assistant treasurer of the Society, the secretaries of the district societies, the chairmen of all standing committees, the editor of the *New England Journal of Medicine*, the president of the Massachusetts Medical Service, if he be a physician and a member of this Society and the medical director of the Massachusetts Medical Service, if he be a member of this Society.

Chapter IV:

Appointment of Director of Medical Information and Education

Section 8. The Council may appoint and dismiss a Director of Medical Information and Education.

Salaries and Appropriations

Section 9. The Council shall vote the salaries of its officers and employees, shall determine the tenure they may respectively have in their offices, the appropriations for its officers, employees, and committees, and such other appropriations as it deems suitable.

No officer, employee, or committee shall exceed the voted appropriation.

No salary to any officer or employee and no regular appropriation shall be increased except on recommendation of the Committee on Finance and by vote of the Council.

The Treasurer is authorized, on recommendation of the Committee on Finance, to pay such monies as may be necessary in the event of emergency, the existence of which shall be determined by the President.

Chapter VI. Officers

Duties of Secretary

Section 4. The Secretary shall attend all meetings of the Society, the Council and the Executive Committee, and shall record their respective proceedings in separate volumes.

He shall cause to be engrossed and shall sign the diplomas of new fellows, if satisfied that they have met the requirements of Chapter I, and shall issue all diplomas and certificates of fellowship.

He shall notify fellows of votes by the Council or Executive Committee granting permission to retire, to resign, to transfer district membership or to have dues remitted, and of votes depriving them of or reinstating them in the privileges of fellowship.

He shall be *ex officio* secretary of all boards of trial, the Board of Supervising Censors, the Committee on Publications, the Committee on Ethics and Discipline, and the Committee on Membership, and shall keep the records of each in separate volumes. He shall be a member *ex officio* of all other committees without power of vote.

He shall notify members of committees of their appointment and of the duties assigned them. On advice of their chairman or secretary he shall give due notice of the time and place of their several meetings.

He shall have custody of the seal of the Society and of all books, papers, manuscripts, prints and paintings belonging to the Society, except such as are in charge of the Treasurer.

He shall issue notices of the meetings of the Council according to such rules as the Council may adopt. He shall issue to every fellow one month before the annual meeting of the Society a program, listing the time and place of that meeting and of the stated meetings of the Council, the boards of censors for that year, and information concerning the payment of assessments and the distribution of publications; if there are any proposed amendments of the by-laws, he shall provide that each program is accompanied by a copy thereof.

He shall transfer fellows from one district to another under the terms of Chapter III, Section 3, and shall report to the Society at its annual meeting the changes in membership during the year.

He shall conduct official correspondence and shall notify officers and delegates of their appointments and of their duties.

He shall keep a directory of the fellows, and shall publish the same, under the direction of the Committee on Publications, at such intervals as may be determined by the Council. He shall furnish this on request to fellows not in arrears.

He shall have jurisdiction over the work of the Executive Secretary and over the work of the Director of Medical Information and Education.

He shall perform such other duties as the Society or the Council may require.

Duties of Executive Secretary

Section 7. The Executive Secretary, under the jurisdiction of the Secretary, shall assist the officers, the Council and such committees as may request his services. He shall also serve as manager of the general office, and shall help in the arrangement of the annual meeting and of such other meetings as are sponsored in whole or in part by the Society.

He shall hold office at the pleasure of the Executive Committee.

Duties of Director of Medical Information and Education

Section 8. The Director of Medical Information and Education, under the jurisdiction of the Secretary, shall promote, in an ethical manner, the educational usefulness of the Society to the fellows; to all licensed physicians in Massachusetts and to the public. He shall also assist the officers, the Council and such District Societies or committees as may request his services.

Expenses of Officers, Executive Secretary, Director of Medical Information and Education, and Committees

Section 9. The traveling and incidental expenses of the officers, of the Executive Secretary, of the Director of Medical Information and Education, of the committees of the Society elected by districts, and of other committees of the Society, on request, shall be paid by the Treasurer, on presentation of an itemized bill duly approved by the President.

These amendments were favorably acted upon by the Executive Committee of the Council on April 7, 1948.

APPENDIX NO. 2

Officers for 1948–1949

President: Daniel B. Reardon, Quincy, 1186 Hancock St.
President-Elect: Arthur W. Allen, Boston, 266 Beacon St.
Vice-President: Donald Munro, Milton. Office, Boston, Boston City Hospital.
Secretary: H. Quimby Gallupe, Waltham. Office, Boston, 8 Fenway.
Treasurer: Eliot Hubbard, Jr., Cambridge, 29 Highland St.
Assistant Treasurer: Norman A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Ave.
Orator: C. Sidney Burwell, Brookline. Office, Boston, 25 Shattuck St.

Committees Elected by the Districts

Executive Committee of the Council — Established 1941 (members *ex-officiis* and one councilor and alternate elected by the councilors of each district medical society).
President: Daniel B. Reardon, Quincy, 1186 Hancock St.
President-Elect: Arthur W. Allen, Boston, 266 Beacon St.

VICE-PRESIDENT: Donald Munro, Milton. Office, Boston, Boston City Hospital.
SECRETARY: H. Quimby Gallupe, Waltham. Office, Boston, 8 Fenway.
TREASURER: Eliot Hubbard, Jr., Cambridge, 29 Highland St.

Term Expires 1949

BARNSTABLE: Paul M. Butterfield, Harwich. (Alternate: Paul P. Henson, Hyannis, 149 Main St.)
BRISTOL NORTH: Joseph L. Murphy, Taunton, 23 Cedar St. (Alternate: Curtis B. Kingsbury, Taunton, 63 Prospect St.)
BRISTOL SOUTH: Richard B. Butler, Fall River, 278 North Main St. (Alternate: Curtis C. Tripp, New Bedford, 416 County St.)
ESSEX NORTH: Rolf C. Norris, Methuen, 247 Broadway. (Alternate: Arnold P. George, Haverhill, 32 Summer St.)
MIDDLESEX EAST: Kenneth L. Maclachlan, Melrose, 1 Bellevue Ave. (Alternate: Justin L. Anderson, Reading, 53 Woburn St.)
PLYMOUTH: George A. Moore, Brockton, 167 Newbury St. (Alternate: John C. Angley, Bryantville, School St.)

Term Expires 1950

BERKSHIRE: John Hughes, Pittsfield, 74 North St. (Alternate: Edward Wyman, Great Barrington, 244 Main St.)
FRANKLIN: Lawrence R. Dame, Greenfield, 78 Federal St. (Alternate: Frank W. Dean, East Northfield, 185 Main St.)
HAMPDEN: Archibald J. Douglas, Westfield, 30 Court St. (Alternate: Frederic Hagler, Springfield, 20 Maple St.)
MIDDLESEX NORTH: William M. Collins, Lowell, 174 Central St. (Alternate: Artemas J. Stewart, Lowell, 310 Merrimack St.)
NORFOLK: Charles J. E. Kickham, Jamaica Plain. Office, Brookline, 1101 Beacon St. (Alternate: Carl Bearse, Boston, 483 Beacon St.)
WORCESTER NORTH: John J. Curley, Leominster, 89 West St. (Alternate: George P. Keaveny, Fitchburg, 62 Fox St.)

Term Expires 1951

ESSEX SOUTH: Albert E. Parkhurst, Beverly, 1 Monument Sq. (Alternate: Edwin D. Reynolds, Danvers, 48 High St.)
HAMPSHIRE: Maurice T. Kennedy, Hadley, 11 Middle St. (Alternate: L. Beverley Pond, Easthampton, 115 Main St.)
MIDDLESEX SOUTH: Joseph C. Merriam, Framingham, 198 Union Ave. (Alternate: John F. Casey, Brighton. Office, Boston, 475 Commonwealth Ave.)
NORFOLK SOUTH: Robert L. Cook, Quincy, 1245 Hancock St. (Alternate: William R. Helfrich, Quincy, 272 Southern Artery.)
SUFFOLK: Harvey A. Kelly, Winthrop, 200 Pleasant St. (Alternate: William E. Browne, Boston, 587 Beacon St.)
WORCESTER: John Fallon, Worcester, 10 Institute Rd. (Alternate: Nicholas S. Scarcello, Worcester, 1 Sheldon St.)

Committee on Nominations — Established 1874 (one councilor and alternate elected yearly by each district medical society).

BARNSTABLE: Paul M. Butterfield, Harwich. (Alternate: Harold F. Rowley, Harwichport.)
BERKSHIRE: Helen Scoville, Pittsfield, House of Mercy Hospital. (Alternate: Charles T. Leslie, Pittsfield, 18 Bank Row.)
BRISTOL NORTH: Joseph L. Murphy, Taunton, 23 Cedar St. (Alternate: Curtis B. Kingsbury, Taunton, 63 Prospect St.)
BRISTOL SOUTH: Henry Wardle, Fall River, 173 Purchase St. (Alternate: Donald R. Mills, Edgartown, Box 654.)
ESSEX NORTH: Charles F. Warren, Amesbury, 155 Main St. (Alternate: Percy J. Look, Andover, 115 Main St.)
ESSEX SOUTH: Peer P. Johnson, Beverly, 1 Monument Sq. (Alternate: Olin S. Pettingill, Middleton, Essex Sanatorium.)
FRANKLIN: Warren D. Thomas, Montague, Central St. (Alternate: Kenneth H. Rice, South Deerfield, 141 Main St.)
HAMPDEN: George L. Schadt, Springfield, 44 Chestnut St. (Alternate: George L. Steele, Springfield, 20 Maple St.)
HAMPSHIRE: Joseph R. Hobbs, Williamsburg, Main St. (Alternate: L. Beverley Pond, Easthampton, 115 Main St.)
MIDDLESEX EAST: Edward M. Halligan, Reading, 37 Salem St. (Alternate: John M. Wilcox, Woburn, 6 Bennett St.)
MIDDLESEX NORTH: C. Stoyle Baker, Lowell, 8 Merrimack St. (Alternate: William M. Collins, Lowell, 174 Central St.)
MIDDLESEX SOUTH: Harold G. Giddings, Newton Centre. Office, Boston, 270 Commonwealth Ave. (Alternate: Fred R. Jouett, Cambridge, 1 Craigie St.)
NORFOLK: Albert Ehrenfried, Boston, 520 Beacon St. (Alternate: Carleton E. Allard, Dorchester, 428 Columbia Rd.)
NORFOLK SOUTH: Henry A. Robinson, Hingham, 205 North St. (Alternate: William R. Helfrich, Quincy, 272 Southern Artery.)
PLYMOUTH: Bradford H. Peirce, South Hanson, Plymouth County Hospital. (Alternate: Alfred L. Duncombe, Brockton, 38 Winthrop St.)
SUFFOLK: Albert A. Hornor, Boston, 319 Longwood Ave. (Alternate: Conrad Wesselhoeft, Boston, 315 Marlboro St.)
WORCESTER: Frank B. Carr, Worcester, 27 Elm St. (Alternate: George W. Ballantyne, Worcester, 27 Elm St.)
WORCESTER NORTH: John J. Curley, Leominster, 89 West St. (Alternate: James V. McHugh, Leominster, 55 West St.)

Committee on Public Relations — Established 1939 (one councilor elected yearly by each district medical society; the president and president-elect of the Society are chairman and vice-chairman, respectively; and the vice-president and secretary of the Society are members ex-officiis).

BARNSTABLE: Paul P. Henson, Hyannis, 149 Main St.
BERKSHIRE: Patrick J. Sullivan, Dalton, 417 Main St.
BRISTOL NORTH: Milton E. Johnson, Attleboro, 33 Bank St.
BRISTOL SOUTH: Harold E. Perry, New Bedford, 159 Cottage St.
ESSEX NORTH: Harold R. Kurth, Lawrence, 57 Jackson St.
ESSEX SOUTH: Bernard Appel, Lynn, 281 Ocean St.
FRANKLIN: John E. Moran, Greenfield, 31 Federal St.
HAMPDEN: Frederic Hagler, Springfield, 20 Maple St.
HAMPSHIRE: Joseph R. Hobbs, Williamsburg, Main St.
MIDDLESEX EAST: Milton J. Quinn, Winchester, 44 Church St.
MIDDLESEX NORTH: Samuel A. Dibbins, Lowell, 528 Andover St.
MIDDLESEX SOUTH: Ralph H. Wells, Lexington, 1430 Massachusetts Ave.
NORFOLK: Dean S. Luce, Canton, 553 Washington St.
NORFOLK SOUTH: Henry A. Robinson, Hingham, 205 North St.
PLYMOUTH: Charles D. McCann, Brockton, 12 Cottage St.
SUFFOLK: Howard F. Root, Boston, 81 Bay State Rd.
WORCESTER: Nicholas S. Scarcello, Worcester, 1 Sheldon St.
WORCESTER NORTH: James V. McHugh, Leominster, 55 West St.

Subcommittees of Committee on Public Relations

TAX-SUPPORTED MEDICAL CARE — Established 1940.
Thomas Hunter, Worcester, *chairman*; Frederick S. Hopkins, Hampden; Albert A. Hornor, Suffolk; Frank W. Snow, Essex North.

COMMITTEE TO MEET WITH THE MEDICAL ADVISORY COMMITTEE OF THE INDUSTRIAL ACCIDENT BOARD — Established 1942.
Gordon M. Morrison, Middlesex South, *chairman*; Charles H. Bradford, Suffolk; Joseph H. Burnett, Middlesex South; Somers Fraser, Suffolk; William W. Teahan, Hampden.

POSTPAYMENT MEDICAL CARE — Established 1942.
Norman A. Welch, Norfolk, *chairman*; Fred A. Bartlett, Norfolk South; Michael F. Barrett, Plymouth; James H. Brewster, Bristol North; James T. Brosnan, Worcester; Lucien R. Chaput, Essex North; Charles F. Fasce, Berkshire; Patrick E. Gear, Hampden; Loring Grimes, Essex South; Francis T. Jantzen, Suffolk; Egon E. Kattwinkel, Middlesex South; Howard M. Kemp, Franklin; William B. LeBrecht, Worcester North; Wilfred L. McKenzie, Middlesex East; Harold E. Perry,

Bristol South; Henry A. Robinson, Norfolk South; Harold F. Rowley, Barnstable; Elmer E. Thomas, Hampshire.

Committee on Legislation — Established 1942 (one councilor elected yearly by each district medical society).

BARNSTABLE: Julius G. Kelley, Pocasset, Barnstable County Sanatorium.
BERKSHIRE: John Hughes, Pittsfield, 74 North St.
BRISTOL NORTH: William M. Stobbs, Attleboro, 63 Bank St.
BRISTOL SOUTH: Curtis C. Tripp, New Bedford, 416 County St.
ESSEX NORTH: Nicandro F. DeCesare, Lawrence, 57 Jackson St.
ESSEX SOUTH: Loring Grimes, Swampscott, 84 Humphrey St.
FRANKLIN: Harold R. Mahar, Orange, 1 High St.
HAMPDEN: Arthur H. Riordan, Indian Orchard, 147 Oak St.
HAMPSHIRE: Justin E. Hayes, Northampton, 16 Centre St.
MIDDLESEX EAST: Justin L. Anderson, Reading, 53 Woburn St.
MIDDLESEX NORTH: Joseph D. Sweeney, Lowell, 174 Central St.
MIDDLESEX SOUTH: Kenneth J. Tillotson, Waverley, McLean Hospital.
NORFOLK: Solomon L. Skvirsky, Boston, 545 State House.
NORFOLK SOUTH: David L. Belding, Hingham, 215 Main St.
PLYMOUTH: Alfred L. Duncombe, Brockton, 38 Winthrop St.
SUFFOLK: William E. Browne, Boston, 587 Beacon St.
WORCESTER: John B. Butts, Worcester, 24 Franklin St.
WORCESTER NORTH: C. Bertram Gay, Fitchburg, 62 Day St.

Subcommittee of the Committee on Legislation

NATIONAL LEGISLATION — Established 1946.
Elmer S. Bagnall, Essex North, *chairman*; David L. Belding, Norfolk South; Vlado A. Getting, Middlesex South; Donald Munro, Suffolk; Augustus Thorndike, Suffolk; Charles G. Hayden, Norfolk.

STANDING COMMITTEES FOR 1948–1949

ELECTED BY THE COUNCIL MAY 24, 1948.

	Date of Appointment
Publications — Established 1825.	
Richard M. Smith, Suffolk	June 6, 1933 (appointed chairman May 21, 1941)
Oliver Cope, Middlesex South	May 21, 1941
John Fallon, Worcester	November 14, 1944
James P. O'Hare, Suffolk	June 9, 1936
Conrad Wesselhoeft, Suffolk	June 2, 1937
Arrangements — Established 1849.	
Harold G. Giddings, Middlesex South	May 22, 1944 (appointed chairman May 24, 1948)
Franklin G. Balch, Jr., Suffolk	May 19, 1947
Gordon A. Donaldson, Middlesex South	May 19, 1947
Albert Ehrenfried, Norfolk	May 24, 1948
John W. Norcross, Middlesex South	May 19, 1947
Ethics and Discipline — Established 1871.	
Ralph R. Stratton, Middlesex East	June 9, 1936 (appointed chairman May 21, 1941)
William J. Brickley, Suffolk	February 3, 1937
Archibald R. Gardner, Middlesex North	May 21, 1941
Fred R. Jouett, Middlesex South	May 21, 1940
Allen G. Rice, Hampden	June 1, 1938
Medical Education — Established 1881.	
Chester S. Keefer, Suffolk	February 4, 1942 (appointed chairman May 19, 1947)
James M. Faulkner, Norfolk	May 21, 1946
George D. Henderson, Hampden	June 1, 1938
Isaac R. Jankelson, Norfolk	May 25, 1942
Membership — Established 1897.	
Lewis S. Pilcher, Middlesex South	July 26, 1946 (appointed chairman May 24, 1948)
William A. R. Chapin, Hampden	May 23, 1945
Peirce H. Leavitt, Plymouth	June 1, 1938
Francis P. McCarthy, Norfolk	May 24, 1948
Samuel N. Vose, Suffolk	March 15, 1944
(John E. Moran, Franklin; Bancroft C. Wheeler, Worcester; Kathleyne S. Snow, Norfolk; — representing the Supervising Censors)	
Public Health — Established 1912.	
Roy J. Ward, Worcester	May 22, 1944 (chairman)
John J. Poutas, Middlesex South	May 21, 1946
Warren R. Sisson, Suffolk	May 19, 1947
Lawrence J. Smith, Hampden	May 19, 1947
Conrad Wesselhoeft, Suffolk	July 27, 1944
Subcommittee of the Committee on Public Health	
MENTAL HEALTH — Established 1947.	
Walter E. Barton, Norfolk	February 5, 1947 (chairman)
William Malamud, Worcester	February 5, 1947
Henry A. Tadgell, Hampshire	February 5, 1947
Medical Defense — Established 1927.	
Horatio Rogers, Suffolk	June 7, 1939 (appointed chairman May 19, 1947)
Edwin D. Gardner, Bristol South	June 7, 1929
Charles J. Kickham, Norfolk	May 21, 1946
John E. Moran, Franklin	May 19, 1947
William R. Morrison, Suffolk	June 9, 1936
Finance — Established 1938.	
Robert W. Buck, Middlesex South	May 21, 1946 (chairman)
Francis C. Hall, Suffolk	July 8, 1943
Fabyan Packard, Middlesex South	May 21, 1946
Bancroft C. Wheeler, Worcester	May 21, 1946
Charles F. Wilinsky, Suffolk	June 2, 1938
Society Headquarters — Established 1942.	
Frank R. Ober, Suffolk	May 22, 1944 (appointed chairman November 1, 1944)
Albert A. Hornor, Suffolk	November 6, 1944
Walter G. Phippen, Essex South	May 21, 1946
George L. Steele, Hampden	May 19, 1947
Industrial Health — Established 1942.	
Daniel L. Lynch, Norfolk	May 25, 1942 (appointed chairman May 21, 1946)
Joseph C. Aub, Suffolk	May 25, 1942
Louis R. Daniels, Middlesex South	May 22, 1944
John G. Downing, Middlesex South	May 22, 1944
Harold R. Kurth, Essex North	May 23, 1945
Frederic N. Manley, Norfolk South	May 19, 1947
Henry C. Marble, Suffolk	May 19, 1947

Advisory Committee to Committee on Industrial Health — Established 1942.

Philip Drinker
Harriet L. Hardy
Emma S. Tousant

Special Committees for 1948–1949

Elected by the Council, May 24, 1948

Cancer — Established 1947.

Shields Warren, Suffolk, *chairman*; Thomas J. Anglem, Suffolk; Ernest M. Daland, Suffolk; Allen G. Rice, Hampden; Channing C. Simmons, Suffolk.

To Meet with the Massachusetts Hospital Association — Established 1940.

Albert E. Parkhurst, Essex South, *chairman*; Edward A. Adams, Worcester North; Edwin D. Gardner, Bristol South; Frederic Hagler, Hampden; Justin E. Hayes, Hampshire; Nicholas S. Scarcello, Worcester; Leland S. McKittrick, Suffolk.

Maternal Welfare — Established 1941.

Duncan Reid, Middlesex South, *chairman*; James M. Baty, Middlesex South; Arthur F. G. Edgelow, Hampden; Samuel B. Kirkwood, Middlesex East; Florence L. McKay, Suffolk; Louis E. Phaneuf, Suffolk; Raymond S. Titus, Norfolk.

Rehabilitation — Established 1941.

Joseph H. Shortell, Suffolk, *chairman*; Benjamin F. Andrews, Worcester; Ralph M. Chambers, Bristol North; William M. Collins, Middlesex North; James J. Regan, Suffolk; Arthur L. Watkins, Middlesex South.

Council Rules — Established 1944.

Charles E. Mongan, Middlesex South, *chairman*; Elmer S. Bagnall, Essex North; Frank R. Ober, Suffolk; Albert A. Hornor, Suffolk.

Postgraduate Medical Education — Established 1944.

W. Richard Ohler, Norfolk, *chairman*; George A. Buckley, Plymouth; Vlado A. Getting, Middlesex South; Robert H. Goodwin, Bristol South; Lewis M. Hurxthal, Suffolk; George P. Keaveny, Worcester North; Leo F. King, Middlesex North; Charles G. Mixter, Suffolk; Joseph W. O'Connor, Worcester; Samuel H. Proger, Norfolk; George S. Reynolds, Berkshire; James L. Smead, Hampden; Harry C. Solomon, Suffolk; Henry D. Stebbins, Essex South; John F. Conlin, Suffolk; Claude E. Welch, Middlesex South.

Medical Economics — Established 1944.

Leland S. McKittrick, Suffolk, *chairman*; Elmer S. Bagnall, Essex North; Allan M. Butler, Suffolk; Vlado A. Getting, Middlesex South; Merrill C. Sosman, Suffolk.

Physical Medicine — Established 1945.

Arthur L. Watkins, Middlesex South, *chairman*; Alexander P. Aitken, Middlesex East; Ralph M. Chambers, Bristol North; Franklin P. Lowry, Middlesex South; Henry A. Tadgell, Hampshire.

To Make Recommendations as to Future Directors of Blue Shield — Established 1945.

Leland S. McKittrick, Suffolk, *chairman* (term expires 1949); Harold G. Giddings, Middlesex South (term expires 1951); Elliott P. Joslin, Suffolk (term expires 1952); Peirce H. Leavitt, Plymouth (term expires 1950).

To Meet with General Hawley with View of Formulating Program in Massachusetts for Medical Care of Veterans and Dependents — Established 1945.

Humphrey L. McCarthy, Norfolk, *chairman*; James K. Bragger, Norfolk; Allen S. Johnson, Hampden.

To Assist the Council on Medical Education and Hospitals of the American Medical Association in the Provisional Approval of Certain Massachusetts Hospitals — Established 1946.

Robert T. Monroe, Norfolk, *chairman*; Laurence D. Chapin, Hampden; H. Quimby Gallupe, Middlesex South; Walter G. Phippen, Essex South; Charles F. Wilinsky, Suffolk.

Postgraduate Assembly — Established 1946.

Leroy E. Parkins, Suffolk, *chairman*; Harold G. Giddings, Middlesex South; Frederick S. Hopkins, Hampden; Charles J. Kickham, Norfolk, Joseph Garland, Suffolk; John F. Conlin, Suffolk.

To Study Income Level for Blue Shield — Established 1946.

Charles F. Wilinsky, Suffolk, *chairman*; Norman H. Bruce, Middlesex South; Raoul L. Drapeau, Middlesex North; Henry L. Kirkendall, Worcester; John W. Spellman, Norfolk.

School Health — Established 1947.

Ernest Morris, Middlesex South, *chairman*; Elmer S. Bagnall, Essex North; Florence L. McKay, Suffolk; Joseph Garland, Suffolk; James O. Wails, Worcester; Thomas F. Reilly, Hampden; Stewart Clifford, Middlesex South; Kenneth L. Maclachlan, Middlesex East.

Auditing

Howard B. Jackson, Norfolk, *chairman*; Frank T. Downey, Middlesex South.

Advisory Council to Women's Auxiliary — Established 1948.

John F. Conlin, Suffolk; *chairman*; Milton J. Quinn, Middlesex East; David L. Belding, Norfolk South.

Advisory Committee for Red Cross Blood Bank — Established 1948.

John F. Conlin, Suffolk, *chairman*; F. Harold Allen, Jr., Middlesex South; William B. Castle, Suffolk; Stephen Brown, Hampshire; William P. Murphy, Suffolk; Joseph F. Ross, Suffolk; C. Stuart Welch, Norfolk.

Advisory Committee on Malpractice Insurance — Established 1948.

Carl Bearse, Norfolk, *chairman*; William J. Brickley, Suffolk; Maurice Fremont-Smith, Suffolk; Charles D. McCann, Plymouth; Horatio Rogers, Suffolk.

Emergency Medical Service — Established 1948.

Reginald Fitz, Suffolk, *chairman*; Donald E. Currier, Suffolk; Charles H. Bradford, Suffolk; Eugene C. Eppinger, Norfolk; J. Roswell Gallagher, Essex North; Allen Johnson, Hampden; Thomas Lanman, Suffolk; Edward D. Churchill, Middlesex South.

Benevolence — Established 1948.

Dwight O'Hara, Middlesex South, *chairman*; Theodore L. Badger, Norfolk; Robert W. Buck, Middlesex South; Eliot Hubbard, Jr., Middlesex South; Charles C. Lund, Suffolk.

Representatives to the Massachusetts Central Health Council.

Elmer S. Bagnall, Essex North, *chairman*; James W. Bunce, Berkshire; Merrill E. Champion, Suffolk; Earle M. Chapman, Suffolk; Roy J. Ward, Worcester; Paul Nathan, Hampden.

Representatives to the Hospital Presidents Association.

H. Quimby Gallupe, Middlesex South; John W. Spellman, Norfolk.

Representative on the Legislative Committee of the Massachusetts Central Health Council.

John F. Conlin, Suffolk.

Representative on a Professional Advisory Committee Organized by the Division of Vocational Rehabilitation of the State Department of Education for the Purpose of Establishing a Program of Physical Restoration.

Joseph H. Shortell, Suffolk.

Representative for Survey by Academy of Pediatrics.

Gerald N. Hoeffel, Middlesex South.

Representatives to the Council of the New England State Medical Societies.

Archibald J. Douglas, Hampden; Gerald N. Hoeffel, Middlesex South; Norman A. Welch, Norfolk.

Twenty-Five Voting Members in Massachusetts Hospital Service, Inc.

Richard Butler, Bristol South; Laurence D. Chapin, Hampden; Lucien R. Chaput, Essex North; Henry W. Godfrey, Middlesex South; Roy J. Heffernan, Norfolk; Albert A. Hornor, Suffolk; Harold R. Kurth, Essex North; John H. Lambert, Middlesex North; Alexander A. Levi, Middlesex South; Joseph C. Merriam, Middlesex South; Albert M. Moloney, Norfolk; Donald Munro, Suffolk; Donald A. Nickerson, Essex South; James P. O'Hare, Suffolk; Albert E. Parkhurst, Essex South; Bradford H. Peirce, Plymouth; Lewis S. Pilcher, Suffolk; Helen S. Pittman, Suffolk; Allen G. Rice, Hampden; Arthur T. Roman, Norfolk; Walter L. Sargent, Norfolk South; Milton J. Schlesinger, Norfolk; George L. Steele, Hampden; Ralph R. Stratton, Middlesex East; and Sidney C. Wiggin, Suffolk.

Delegates and Alternates to the House of Delegates of the American Medical Association for 1948–1949

Delegates	Alternates
June 1, 1947 to June 1, 1949	
Charles J. Kickham, Norfolk	John Fallon, Worcester
Leland S. McKittrick, Suffolk	Harold R. Kurth, Essex North
June 1, 1948 to June 1, 1950	
Patrick J. Sullivan, Berkshire	Nahum R. Pillsbury, Norfolk South
John J. Curley, Worcester North	Patrick E. Gear, Hampden
Walter G. Phippen, Essex South	John I. B. Vail, Barnstable
Frank W. Snow, Essex North	Frederick Hinchliffe, Norfolk South

Councilors for 1948–1949

(Elected by the District Medical Societies at Their Annual Meetings, April 15 to May 15, 1948.)

Barnstable

H. P. Hopkins, Chatham, V. P.
P. M. Butterfield, Harwich, A. E. C., M. N. C.
P. P. Henson, Hyannis, 149 Main St., E. C., P. R. C.
J. G. Kelley, Pocasset, Barnstable County Sanatorium, Leg. C.
H. F. Rowley, Harwichport, A. M. N. C.
Frederick Sanborn, Osterville, Bates Ave., Sec.

Berkshire

G. S. Reynolds, Pittsfield, 100 North St., V. P.
D. N. Beers, Pittsfield, 74 North St., Sec.
Modestino Criscitiello, Pittsfield, 28 North St.
Antoine Dumouchel, North Adams, 56 Summer St., A. E. C.
John Hughes, Pittsfield, 74 North St., Leg. C.
C. T. Leslie, Pittsfield, 18 Bank Row, A. M. N. C.
Helen M. Scoville, Pittsfield, House of Mercy Hospital, M. N. C., E. C.
P. J. Sullivan, Dalton, 471 Main St., P. R. C.
E. R. Wyman, Great Barrington, 259 Main St.

Bristol North

J. V. Chatigny, Taunton, 43 West Britannia St., V. P.
W. E. Dawson, Taunton, 58 Winthrop St., Sec.
M. E. Johnson, Attleboro, 33 Bank St., P.R.C.
C. B. Kingsbury, Taunton, 63 Prospect St., A. E. C., A. M. N. C.
J. L. Murphy, Taunton, 23 Cedar St., E. C., M. N. C.
W. M. Stobbs, Attleboro, 63 Bank St., Leg. C.

Bristol South

A. J. Pothier, New Bedford, 720 County St., V. P.
R. B. Butler, Fall River, 278 North Main St., E. C.
J. C. Corrigan, Fall River, 422 North Main St.
J. E. Fell, Fall River, 181 Purchase St., Sec.
J. A. Fournier, Fall River, 11 Choate St.
D. F. Gallery, Fall River, 151 Rock St.
E. D. Gardner, Marion, Box 175.
R. H. Goodwin, New Bedford, 15 South 6th St.
William Mason, Fall River, 151 Rock St.
D. R. Mills, Edgartown, Pease Point Way, A. M. N. C.
H. E. Perry, New Bedford, 159 Cottage St., P. R. C.
C. C. Persons, New Bedford, 118 Cottage St.
C. C. Tripp, New Bedford, 416 County St., A. E. C., Leg. C.
Henry Wardle, Fall River, 173 Purchase St., M. N. C.

Essex North

R. E. Blais, Amesbury, 165 Main St., V. P.
M. F. Ames, Newburyport, P. O. Box 88.
E. S. Bagnall, Groveland, 281 Main St., Ex-Pres.
R. V. Baketel, Methuen, 7 Hampshire St.
J. A. Bradley, Methuen, 103 Lowell St.
N. F. DeCesare, Lawrence, 57 Jackson St., Leg. C.
A. P. George, Haverhill, 32 Summer St., A. E. C.
S. P. Humphreys, Newbury, 155 High Rd.
P. J. Look, Andover, 115 Main St., A. M. N. C.
H. R. Kurth, Lawrence, 57 Jackson St., Sec., P. R. C.
R. C. Norris, Methuen, 247 Broadway, E. C.
F. W. Snow, Newburyport, 24 Essex St.
L. T. Stokes, Haverhill, 30 Summer St.
F. N. Sweetsir, Merrimac, 19 Main St.
C. F. Warren, Amesbury, 1 School St., M. N. C.
C. A. Weiss, Lawrence, 160 Jackson St.

Essex South

E. L. Peirson, Salem, 374 Essex St., V. P.
Bernard Appel, Lynn, 281 Ocean St., P. R. C.
W. W. Babson, Gloucester, 79 Prospect St.
R. E. Foss, Peabody, 125 Main St.
S. N. Gardner, Salem, 24 Chestnut St.
Loring Grimes, Swampscott, 84 Humphrey St., Leg. C.
R. P. Hallett, Gloucester, Beach Rd.
C. A. Herrick, Manchester, 21 Union St.
P. P. Johnson, Beverly, 1 Monument Sq., M. N. C.
R. T. Moulton, Salem, 37 Warren St.
A. E. Parkhurst, Beverly, 1 Monument Sq., E. C.
O. S. Pettingill, Middleton, Essex Sanatorium, A. M. N. C.
Walter G. Phippen, Salem, 31 Chestnut St., Ex-Pres.
E. D. Reynolds, Danvers, 48 High St., A. E. C.
H. D. Stebbins, Salem, 342 Essex St., Sec.
P. E. Tivnan, Salem, 70 Washington St.
C. F. Twomey, East Lynn, 80 Ocean St.
R. J. Williams, Lynn, 39 Lynn Shore Drive.

Franklin

L. R. Dame, Greenfield, 78 Federal St., V. P., E. C.
F. W. Dean, East Northfield, 185 Main St., A. E. C.
H. R. Mahar, Orange, 1 High St., Leg. C.
J. E. Moran, Greenfield, 31 Federal St., P. R. C.
K. H. Rice, South Deerfield, 141 Main St., A. M. N. C.
M. M. Sisson, Greenfield, 31 Federal St., Sec.
W. D. Thomas, Montague, Central St., M. N. C.

Hampden

R. D. Hildreth, Westfield, 90 Elm St., V. P.
F. H. Allen, Holyoke, 16 Fairfield St.
E. P. Bagg, Holyoke, 207 Elm St.. Ex-Pres.
R. L. Barrett, Springfield, 21 Maple St.
J. M. Birnie, Springfield, 146 Chestnut St., Ex-Pres.
W. A. R. Chapin, Springfield, 121 Chestnut St.
J. L. Chereskin, Springfield, 333 Bridge St.
G. B. Corcoran, West Springfield, 84 Park St.
A. J. Douglas, Westfield, 30 Court St., E. C.
E. C. Dubois, Springfield, 174 Buckingham St.
A. F. G. Edgelow, Springfield, 76 Maple St.
Adolph Franz, Jr.. Holyoke, 1158 Northampton St.
P. E. Gear, Holyoke, 188 Chestnut St.
J. M. Gilchrist, Springfield, 121 Chestnut St.
Frederic Hagler, Springfield, 20 Maple St.. A. E. C., P. R. C.
F. S. Hopkins, Springfield, 146 Chestnut St.
F. D. Jones, Springfield, 20 Maple St.
Charles Jurist, Springfield, 70 Chestnut St.
John Pallo, Westfield, 97 Elm St.
A. G. Rice, Springfield, 146 Chestnut St.
A. H. Riordan, Indian Orchard, 147 Oak St., Leg. C.
G. L. Schadt, Springfield, 44 Chestnut St., M. N. C., Ex-Pres.
J. A. Seaman, Springfield, 20 Maple St.
G. L. Steele, Springfield, 20 Maple St., A. M. N. C.
W. W. Williams, Springfield, 30 Magnolia Terrace, Sec.

Hampshire

E. J. Manwell, Northampton, 16 Centre St., V. P.
R. S. Clapp, Amherst, 110 North Pleasant St.
J. E. Hayes, Northampton, 16 Centre St., Leg. C.
J. R. Hobbs, Williamsburg, Main St., P. R. C., M. N. C.
M. T. Kennedy, Hadley, 11 Middle St., E. C.
J. G. Pekala, Northampton, 245 Main St.
L. B. Pond, Easthampton, 115 Main St., A. E. C., A. M. N. C.
F. Mary P. Snook, Worthington, Sec.

Middlesex East

M. J. Quinn, Winchester, 44 Church St., V. P., P. R. C.
J. L. Anderson, Reading, 53 Woburn St., Leg. C., A. E.C.
J. W. Burke, Wakefield, 18 Lafayette St.
T. P. Devlin, Stoneham, 38 Pleasant St.
Robert Dutton, Wakefield, 33 Avon St.
E. M. Halligan, Reading, 37 Salem St., M. N. C.
R. W. Layton, Melrose, 8 Porter St., Sec.
K. L. Maclachlan, Melrose, 1 Bellevue Ave., E. C.
H. L. Mueller, Winchester, 31 Church St.
R. R. Stratton, Melrose, 538 Lynn Fells Parkway, C.
J. M. Wiltox, Woburn, 6 Bennet St., A. M. N. C.

Middlesex North

J. Y. Rodger, Lowell, 226 Central St., V. P.
C. S. Baker, Lowell, 8 Merrimack St., M. N. C.
R. E. Cole, Westford.
W. M. Collins, Lowell, 174 Central St., A. M. N. C., E. C.
S. A. Dibbins, Lowell, 528 Andover St., P. R. C.
L. J. Hall, Lowell, 8 Merrimack St.
L. F. King, Lowell, 308 Merrimack St.
B. D. Leahey, Lowell, 9 Central St., Sec.
A. J. Stewart, Lowell, 310 Merrimack St., A. E. C.
J. D. Sweeney, Lowell, 174 Central St., Leg. C.

Middlesex South

J. F. Casey, Allston. Office, Boston 15, 475 Commonwealth Ave., V. P., A. E. C.
G. G. Bailey, Belmont. Office, Boston 15, 412 Beacon St.
E. W. Barron, Malden. Office, Boston, 20 Ash St.
Harris Bass, Everett 49, 351 Broadway.
J. M. Baty, Belmont. Office, Brookline 46, 1101 Beacon St.
J. D. Bennett, West Somerville 44, 72 College Ave.
W. O. Blanchard, Newton 58, 465 Centre St.
H. K. Bloom, Everett, 834 Broadway.
G. F. H. Bowers, Newton Highlands 61, 156 Woodward St.
Alice M. Broadhurst, Watertown, 259 Mt. Auburn St.
Madelaine R. Brown, Cambridge. Office, Boston 16, 264 Beacon St.
R. N. Brown, Malden, 621 Main St.
R. W. Buck, Waban. Office, Boston 15, 5 Bay State Rd., C.
E. J. Butler, Cambridge, 25 Garden St.
C. W. Clark, Newtonville 60, 363 Walnut St.
E. A. Cooney, Newton. Office, Boston 16, 270 Commonwealth Ave.
Oliver Cope, Cambridge. Office, Boston 14, Massachusetts General Hospital.
W. H. Crosby, Brighton, 304 Faneuil St.
J. A. Daley, Natick, 36 Pond St.
C. L. Derick, Newton Highlands. Office, Boston 15, 412 Beacon St.
J. G. Downing, Newton. Office, Boston 15, 520 Commonwealth Ave.
D. J. Duggan, Malden 48, 3 Hawthorne St.
A. G. Engelbach, Cambridge, 330 Mt. Auburn St.
W. C. Feeley, Cambridge, 859 Massachusetts Ave.
C. W. Finnerty, West Somerville 44, 440 Broadway.
J. M. Flynn, Belmont. Office, Boston 15, 412 Beacon St.
H. Quimby Gallupe, Waltham. Office, Boston 15, 8 Fenway, Secretary.
V. A. Getting, Belmont. Office, Boston 33, 545 State House.
H. G. Giddings, Newton Centre. Office, Boston, 270 Commonwealth Ave., M. N. C., C.
H. W. Godfrey, Auburndale 66, 14 Hancock St.
J. L. Golden, Medford 55, 86 Forest St.
A. D. Guthrie, Medford 55, 408 Salem St.
Eliot Hubbard, Jr., Cambridge, 29 Highland St., Treasurer.
A. M. Jackson, Everett, 512 Broadway.
F. R. Jouett, Cambridge, 1 Craigie St., A. M. N. C.
S. B. Kelley, West Newton, 34 Exeter St.
H. A. Kontoff, Newton Centre. Office, Boston, 479 Beacon St.
J. J. Lepore, Marlboro, 96 West Main St.
A. A. Levi, Newton. Office, Boston 15, 481 Beacon St., Sec.
H. E. MacMahon, Cambridge. Office, Boston, 416 Huntington Ave.
A. N. Makechnie, Cambridge, 14 Upland Rd.
R. A. McCarty, Waltham 54, 465 Lexington St.
J. J. McSweeney, Somerville, 26 Bow St.
J. C. Merriam, Framingham, 198 Union Ave., E. C.
Dudley Merrill, Cambridge, 51 Brattle St.
C. E. Mongan, Somerville, 24 Central St., Ex-Pres.
G. M. Morrison, Waban. Office, Boston 15, 520 Commonwealth Ave., C.
D. G. Nutter, Newton Centre, 1094 Centre St.
Dwight O'Hara, Waltham. Office, Boston 15, 416 Huntington Ave., Ex-Pres.
Fabyan Packard, Belmont, 154 Washington St.
L. G. Paul, Newton Centre. Office, Boston 16, 270 Commonwealth Ave.
L. S. Pilcher, Newton Centre. Office, Boston 15, 12 Bay State Rd., C.
Randolph Piper, Concord, 14 Sudbury Rd.
T. E. Reilly, Marlboro, 6 Newton St.
Max Ritvo, Newton. Office, Boston 15, 416 Marlboro St.
G. A. Saunders, Arlington, 50 Pleasant St.
M. J. Schlesinger, Newton. Office, Boston 15, 330 Brookline Ave.
E. W. Small, Belmont, 68 Leonard St.
H. P. Stevens, Cambridge, 1 Craigie St.
K. J. Tillotson, Waverley, McLean Hospital, Leg. C.
A. B. Toppan, Watertown, 289 Mt. Auburn St.
J. H. Townsend, Belmont, 195 Marsh St.
J. E. Vance, Natick. Office, Boston 15, 29 Bay State Rd.
C. F. Walcott, Cambridge, 81 Sparks St.
A. L. Watkins, Arlington. Office, Boston 14, Massachusetts General Hospital.
R. H. Wells, Lexington, 1430 Massachusetts Ave., P. R. C.
B. M. Wein, Newton. Office, Boston 15, 471 Commonwealth Ave.
Alfred Worcester, Waltham 54, 314 Bacon St., Ex-Pres.
Hovhannes Zovickian, Watertown, 528 Mt. Auburn St.

Norfolk

G. W. Papen, Brookline. Office, Boston 9, 31 Milk St., V. P.
A. A. Abrams, Brookline 46, 153 Dean Rd.
C. E. Allard, Dorchester, 428 Columbia Rd., A. M. N. C.

B. E. Barton, West Roxbury, 10 Richwood St., Sec.
Carl Bearse, Boston, 483 Beacon St., A. E. C.
Elizabeth L. Broyles, Wellesley, Simpson Infirmary.
J. H. Cauley, Dorchester, 8 Carruth St.
G. L. Doherty, West Roxbury. Office, Boston 15, 466 Commonwealth Ave.
Albert Ehrenfried, Brookline. Office, Boston 15, 520 Beacon St., M. N. C.
J. M. Faulkner, Brookline. Office, Boston, 80 East Concord St.
P. S. Foisie, Milton, 65 Hillsview Rd.
Susannah Friedman, Roxbury. Office, Boston 15, 485 Commonwealth Ave.
T. R. Goethals, Brookline, 34 Hawthorn Rd.
D. L. Halbersleben, Brookline 46, 42 Goodnough Rd.
J. A. Halsted, Dedham. Office, Jamaica Plain, Faulkner Hospital.
H. B. Harris, Milton. Office, Dorchester, 487 Columbia Rd.
C. C. G. Hayden, Brookline. Office, Boston, 38 Chauncy St.
R. J. Heffernan, Jamaica Plain. Office, Brookline 46, 1101 Beacon St.
Gilbert Horrax, Brookline. Office, Boston, 605 Commonwealth Ave.
P. J. Jakmauh, Milton. Office, South Boston 27, 509 Broadway.
I. R. Jankelson, Jamaica Plain. Office, Boston 15, 483 Beacon St.
L. F. Johnson, Brookline, Longwood Towers.
C. J. Kickham, Brookline. Office, Boston, 508 Commonwealth Ave.
C. J. E. Kickham, Jamaica Plain. Office, Brookline 46, 1101 Beacon St., E. C.
D. L. Lionberger, Dedham, 709 East St.
D. S. Luce, Canton, 553 Washington St., P. R. C.
C. M. Lydon, Dorchester, 276 Bowdoin St.
D. L. Lynch, Roslindale. Office, Boston, 245 State St., C.
T. F. P. Lyons, Milton. Office, Boston 16, 270 Commonwealth Ave.
F. P. McCarthy, Milton. Office, Boston 15, 371 Commonwealth Ave.
H. L. McCarthy, Brookline. Office, Boston 15, 479 Beacon St.
R. L. Mason, Wellesley Hills, 17 Allen Rd.
R. T. Monroe, Brookline. Office, Boston 16, 270 Commonwealth Ave.
F. J. Moran, Dedham, 395 Washington St.
H. R. Morrison, Brookline. Office, Boston, 370 Marlboro St.
Hyman Morrison, Brookline. Office, Boston 15, 483 Beacon St.
D. J. Mullane, Brookline 46, 1101 Beacon St.
H. A. Novack, Brookline. Office, Boston, 471 Commonwealth Ave.
J. J. O'Connell, Dorchester, 1061 Dorchester Ave.
W. R. Ohler, Jamaica Plain. Office, Boston 15, 319 Longwood Ave.
E. E. O'Neil, Brookline. Office, Boston, 270 Commonwealth Ave.
R. S. Palmer, Brookline. Office, Boston, 330 Dartmouth St.
H. C. Petterson, West Roxbury. Office, Boston 15, 29 Bay State Rd.
S. H. Proger, Brookline. Office, Boston, 30 Bennet St.
H. A. Rice, Canton, 742 Washington St.
S. A. Robins, Boston 15, 636 Beacon St.
D. D. Scannell, Jamaica Plain. Office, Boston 15, 475 Commonwealth Ave.
J. A. Seth, Milton. Office, Boston 15, 47 Bay State Rd.
L. A. Sieracki, Norwood, 71 Winter St.
S. L. Skvirsky, Brighton. Office, Boston, 545 State House, Leg. C.
E. C. Smith, Brookline. Office, Boston 15, 520 Commonwealth Ave.
Kathleyne S. Snow, Jamaica Plain. Office, Boston 15, 466 Commonwealth Ave.
J. W. Spellman, Chestnut Hill. Office, Brookline, 1101 Beacon St.
A. R. Stagg, Medfield, 25 Pleasant St.
W. J. Walton, Dorchester, 106 Bowdoin St.
N. A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Ave., Asst. Treas.
G. F. Wilkins, Brookline. Office, Boston, 245 State St.
P. R. Withington, Milton, 350 Randolph Ave.
Marjorie Woodman, Jamaica Plain. Office, Boston, 21 Bay State Rd.
E. T. Wyman, Brookline. Office, Boston 15, 319 Longwood Ave.

Norfolk South

Frederick Hinchliffe, Cohasset, 117 South Main St., V. P.
F. A. Bartlett, Wollaston 70, 308 Beale St.
D. L. Belding, Hingham. Office, Boston, 80 East Concord St., Leg. C.
Harry Braverman, Quincy 69, 43 School St.
E. M. Britton, Wollaston, 25 Elm Ave.
R. L. Cook, Quincy, 1245 Hancock St.. E. C.
W. R. Helfrich, Quincy, 166 Washington St., A. M. N. C., A. E. C.
E. K. Jenkins, South Braintree, Norfolk County Hospital, Sec.
N. R. Pillsbury, South Braintree, Norfolk County Hospital.
D. B. Reardon, Quincy 69, 1186 Hancock St., President.
H. A. Robinson, Hingham, 205 North St., P. R. C., M. N. C.
R. R. Ryan, East Weymouth, 681 Broad St.
R. G. Vinal, Norwell, Main St.

Plymouth

G. A. Buckley, Brockton, 12 Cottage St., V. P.
J. C. Angley, Bryantville, School St., A. E. C., A. M. N. C.
A. L. Duncombe, Brockton, 38 Winthrop St., Leg. C.
Samuel Gale, Brockton, The Checkerton, Sec.
H. H. Hamilton, Plymouth, 70 Court St.
P. H. Leavitt, Brockton, 129 West Elm St.
C. D. McCann, Brockton, 12 Cottage St., P. R. C.
G. A. Moore, Brockton, 167 Newbury St., E. C.
B. H. Peirce, South Hanson, Plymouth County Hospital, M. N. C.
E. L. Perry, Middleboro, 39 Oak St.

Suffolk

A. J. A. Campbell, Brighton. Office, Boston, 520 Commonwealth Ave., V. P.
H. L. Albright, Boston 15, 412 Beacon St.
A. W. Allen, Boston 15, 264 Beacon St.. President-Elect.
T. J. Anglem, Brookline, 1180 Beacon St.
M. D. Altschule, Boston, 330 Brookline Ave.
J. W. Bartol, Boston, 1 Chestnut St., Ex-Pres.
C. H. Bradford, Boston, 220 Beacon St.
W. J. Brickley, Boston 15, 524 Commonwealth Ave.
W. E. Browne, Boston 15, 587 Beacon St., A. E. C., Leg. C.
A. M. Butler, Boston 14, Massachusetts General Hospital.
E. M. Chapman, Brookline. Office, Boston, 266 Beacon St.
M. H. Clifford, Cambridge. Office, Boston, 501 Boylston St.
A. P. DerHagopian, Chelsea, 39 Cary Ave.
H. H. Faxon, Brookline. Office, Boston, 264 Beacon St.
N. W. Faxon, Boston 14, Massachusetts General Hospital.
Reginald Fitz, Boston 15, 319 Longwood Ave., Ex-Pres.
Maurice Fremont-Smith, Boston 15, 12 Hereford St.
Channing Frothingham, Boston, 101 Bay State Rd., Ex-Pres.
Joseph Garland, Brookline. Office, Boston, 8 Fenway.
G. L. Gately, East Boston, 624 Bennington St.
A. A. Hornor, Boston 15, 319 Longwood Ave., M. N. C.
H. E. Kennard, Newton Centre. Office, Boston, 372 Marlboro St.
C. S. Keefer, Boston, 65 East Newton St., C.
H. A. Kelly, Winthrop, 200 Pleasant St., E. C.
T. H. Lanman, Boston 15, 300 Longwood Ave.
R. I. Lee, Boston 15, 264 Beacon St., Ex-Pres.
C. C. Lund, Boston, 20 Gloucester St.
C. F. Maraldi, Boston, 276 Commonwealth Ave.
L. S. McKittrick, Brookline. Office, Boston, 205 Beacon St.
W. J. Mixter, Chestnut Hill. Office, Boston 15, 319 Longwood Ave.

Donald Munro, Boston 18, 818 Harrison Ave., Vice-President.
H. L. Musgrave, Revere, 620 Beach St.
F. R. Ober, Boston, 234 Marlboro St., Ex-Pres.
F. W. O'Brien, Boston 15, 475 Beacon St.
J. P. O'Hare, Chestnut Hill. Office, Boston, 520 Commonwealth Ave.
L. E. Parkins, Brookline. Office, Boston 15, 12 Bay State Rd., C.
L. E. Phaneuf, Boston 16, 270 Commonwealth Ave.
Helen S. Pittman, Boston 16, 264 Beacon St.
J. H. Pratt, Boston 11, 30 Bennet St.
J. J. Regan, South Boston. Office, Boston, 520 Commonwealth Ave.
W. H. Robey, Boston, 202 Commonwealth Ave., Ex-Pres.
Horatio Rogers, Boston, 264 Beacon St., C.
H. F. Root, Boston, 81 Bay State Rd., P. R. C.
C. G. Shedd, Boston, 422 Beacon St., Sec.
R. M. Smith, Boston, 330 Dartmouth St., C.
C. M. Stearns, Chelsea, 116 Hawthorn St.
Augustus Thorndike, Boston 15, 319 Longwood Ave.
Conrad Wesselhoeft, Boston, 315 Marlboro St. A. M. N. C.
C. F. Wilinsky, Boston, 330 Brookline Ave.

Worcester

J. J. Dumphy, Worcester, 390 Main St., V. P.
A. W. Atwood, Worcester, 390 Main St.
G. W. Ballantyne, Worcester, 27 Elm St., A. M. N. C.
F. P. Bousquet, Worcester, 390 Main St.
J. B. Butts, Worcester, 24 Franklin St., Leg. C.
J. T. B. Carmody, Worcester, 340 Main St.
F. B. Carr, Worcester, 27 Elm St., M. N. C.
E. J. Crane, Holden, Armington Lane.
Paul Dufault, Rutland, Rutland State Sanatorium.
G. R. Dunlop, Worcester, 53 Massachusetts Ave.
John Fallon, Worcester, 10 Institute Rd., E. C.
Donald Hight, Worcester, 27 Elm St., Sec.
Thomas Hunter, Shrewsbury, 545 Main St., C.
H. L. Kirkendall, Worcester, 27 Elm St.
D. G. Ljungberg, Worcester, 36 Pleasant St.
J. A. Lundy, Oxford, 26 Main St.
J. C. McCann, Worcester, 390 Main St.
D. K. McClusky, Worcester, 7 Hawthorne St.
J. W. McKoan, Worcester, 36 Pleasant St.
J. M. Olson, Westboro, 54 West Main St.
F. A. O'Toole, Clinton, 181 Chestnut St.
E. L. Richmond, Worcester, 390 Main St.
N. S. Scarcello, Worcester, 1 Sheldon St., P. R. C., A. E. C.
R. J. Ward, Worcester, 9 Bellevue St., C.
B. C. Wheeler, Worcester, 27 Elm St.

Worcester North

D. B. Cheetham, Athol, 164 Exchange St., V. P.
J. J. Curley, Leominster, 89 West St., E. C., M. N. C.
C. B. Gay, Fitchburg, 62 Day St., Leg. C.
G. P. Keaveny, Fitchburg, 62 Fox St., A. E. C.
J. V. McHugh, Leominster, 55 West St., P. R. C., A. M. N. C.
C. N. McPeak, Fitchburg, 18 Hartwell St.
J. G. Simmons, Fitchburg, 30 Myrtle Ave., Sec.

The initials *E. C.* following the name of a Councilor indicate that he is a member of the Executive Committee, and *A. E. C.* that he is an alternate member of the Executive Committee; *M. N. C.* that he is a member of the Committee on Nominations and *A. M. N. C.* that he is an alternate member of the Committee on Nominations; *Leg. C.* that he is a member of the Committee on Legislation; *P. R. C.* that he is a member of the Committee on Public Relations; *V. P.* that a member is a councilor by virtue of his office as president of a district society and so vice-president of the general society; *C.* by virtue of his office as chairman of a standing committee; *Sec.* by virtue of his office as secretary of a district society and *Ex-Pres.* by virtue of being a past president.

Censors for 1948–1949

Barnstable

P. P. Henson, Hyannis, *supervisor*.
D. H. Hiebert, Provincetown.
D. E. Higgins, Barnstable.
Joseph Kelley, Orleans.
O. S. Simpson, Barnstable.

Berkshire

C. T. Leslie, Pittsfield, *supervisor*.
A. C. England, Pittsfield.
W. T. Frawley, Pittsfield.
A. M. Gangemi, North Adams.
G. S. Wickham, Lee.

Bristol North

J. L. Murphy, Taunton, *supervisor*.
J. H. Brewster, Attleboro.
C. B. Kingsbury, Taunton.
A. J. Leddy, Taunton.
H. G. Vaughan, Attleboro.

Bristol South

Henry Wardle, Fall River, *supervisor*.
F. M. Howes, New Bedford.
W. F. MacKnight, Fall River.
E. A. McCarthy, Fall River.
C. C. Persons, New Bedford.

Essex North

L. C. Peirce, Newburyport, *supervisor*.
H. M. Allen, Lawrence.
A. B. George, Haverhill.
Julius Kay, North Andover.
H. G. Nichols, Haverhill.

Essex South

S. N. Gardner, Salem, *supervisor*.
L. F. Box, Beverly.
W. R. Irving, Gloucester.
J. R. Shaughnessy, Salem.
C. F. Twomey, Lynn.

Franklin

F. J. Barnard, Greenfield, *supervisor*.
John Colleran, South Deerfield.
Wendell Matthews, Shelburne Falls.
John E. Moran, Greenfield.
H. A. Rys, Turners Falls.

Hampden

John Pallo, Westfield, *supervisor*.
Eugene Beauchamp, Springfield.
Alphonso Palermo, Springfield.
Lawrence Putnam, Holyoke.
J. L. Smead, Springfield.

Hampshire

L. B. Pond, Easthampton, *supervisor*.
Stephen Brown, Northampton.
M. E. Cooney, Northampton.
T. F. Corriden, Northampton.
J. E. Hayes, Northampton.

Middlesex East

T. P. Devlin, Stoneham, *supervisor*.
C. R. Baisley, Reading.
H. A. Bouvé, Wakefield.
S. H. Moses, Winchester.
M. H. Rovner, Melrose.

Middlesex North

L. F. King, Lowell, *supervisor*.
Philip Berman, Lowell.
Harry Black, Lowell.
E. H. Latham, Lowell.
Charles Roughan, Lowell.

Middlesex South

J. M. Baty, Belmont, *supervisor*.
W. O. Blanchard, Newton.
Oliver Cope, Cambridge.
H. J. Crumb, Lexington.
E. A. Gaston, Framingham.

Norfolk

K. S. Snow, Jamaica Plain, *supervisor*.
I. R. Jankelson, Jamaica Plain.
W. C. Moloney, Forest Hills.
H. A. Novack, Brookline.
E. E. O'Neil, Chestnut Hill.

Norfolk South
F. A. Bartlett, Wollaston, *supervisor*.
Arthur Rappeport, Quincy.
H. S. Reid, Cohasset.
R. E. Ross, South Braintree.
W. L. Sargent, Quincy.

Plymouth
E. L. Perry, Middleboro, *supervisor*.
L. A. Alley, Lakeville.
Jacob Brenner, North Easton.
E. B. Gilmore, Brockton.
R. E. Swenson, Plymouth.

Suffolk
J. H. Pratt, Boston, *supervisor*.
J. F. Collins, Revere.
R. L. Goodale, Boston.
L. M. Hurxthal, Boston.
J. J. Todd, Boston.

Worcester
B. C. Wheeler, Worcester, *supervisor*.
J. B. Butts, Worcester.
Thomas Hunter, Shrewsbury.
H. L. Kirkendall, Worcester.
J. W. McKoan, Worcester.

Worcester North
C. B. Gay, Fitchburg, *supervisor*.
K. J. Jolma, Gardner.
J. W. Mason, Ashburnham.
E. R. Pickwick, Fitchburg.
C. A. Wheeler, Leominster.

Vice-Presidents of the Massachusetts Medical Society (*Ex-Officiis*) for 1948–1949

Presidents of District Medical Societies

(Arranged according to seniority of fellowship in the Massachusetts Medical Society)

Norfolk South — Frederick Hinchliffe, Cohasset.
Middlesex South — John F. Casey, Boston.
Plymouth — George A. Buckley, Brockton.
Middlesex North — James Y. Rodger, Lowell.
Norfolk — George W. Papen, Brookline.
Bristol North — Joseph V. Chatigny, Taunton.
Worcester — John J. Dumphy, Worcester.
Bristol South — Aubrey J. Pothier, New Bedford.
Worcester North — Donald B. Cheetham, Athol.
Middlesex East — Milton J. Quinn, Winchester.
Berkshire — George S. Reynolds, Pittsfield.
Suffolk — Alexander J. A. Campbell, Brighton.
Essex North — Robert E. Blais, Amesbury.
Essex South — Edward L. Peirson, Salem.
Franklin — Lawrence R. Dame, Greenfield.
Hampden — Robert D. Hildreth, Westfield.
Barnstable — Henry P. Hopkins, Chatham.
Hampshire — Edward J. Manwell, Northampton.

Commissioners of Trial for 1948–1949

Barnstable — F. O. Cass, Provincetown.
Berkshire — J. W. Bunce, North Adams.
Bristol North — J. W. Cook, Mansfield.
Bristol South — A. C. Lewis, Fall River.
Essex North — R. V. Baketel, Methuen.
Essex South — L. H. Limauro, Lynn.
Franklin — Kenneth Jacobus, Turners Falls.
Hampden — G. D. Henderson, Holyoke.
Hampshire — R. C. Byrne, Hatfield.
Middlesex East — G. R. Murphy, Melrose.
Middlesex North — C. M. Roughan, Lowell.
Middlesex South — H. P. Stevens, Cambridge.
Norfolk — W. J. Walton, Dorchester.
Norfolk South — W. L. Sargent, Quincy.
Plymouth — W. M. Carr, Whitman.
Suffolk — Augustus Thorndike, Chestnut Hill.
Worcester — A. W. Atwood, Worcester.
Worcester North — W. E. Currier, Leominster.

Officers of the Sections for 1948–1949

Medicine
Chairman, Laurence C. Ellis, Cambridge; *vice-chairman*, Allen S. Johnson, Longmeadow; *secretary*, James A. Halsted, Dedham.

Surgery
Chairman, Robert E. Gross, Boston; *secretary*, Franklin G. Balch, Jr., Boston. Executive Committee — J. Hartwell Harrison, Brookline.

Pediatrics
Chairman, W. Bradford Adams, Springfield; *secretary*, Gerald N. Hoeffel, Boston.

Obstetrics and Gynecology
Chairman, James F. Conway, Brookline; *vice-chairman*, Daniel McSweeney, Boston; *secretary*, Duncan E. Reid, Boston.

Radiology
Chairman, Albert B. Maloney, Boston; *secretary*, Lawrence Robbins, Boston.

Physiotherapy
Chairman, David C. Ditmore, Boston; *secretary*, Arthur L. Watkins, Boston.

Dermatology and Syphilology
Chairman, Francis P. McCarthy, Boston; *secretary*, Alfred Hollander, Springfield.

Anesthesiology
Chairman, Morris J. Nicholson, Boston; *secretary*, Jacob Fine, Beverly.

Officers of the District Medical Societies for 1948–1949

Barnstable — *President*, Henry P. Hopkins, Chatham; *vice-president*, Arthur J. D'Elia, Harwichport; *secretary*, Frederick Sanborn, Osterville; *treasurer*, Frank Travers, Barnstable; *librarian*, Carroll H. Keene, Chatham; *executive and public relations councilor*, Paul P. Henson, Hyannis; *legislative councilor*, Julius G. Kelley, Pocasset.

Berkshire — *President*, George S. Reynolds, Pittsfield; *vice-president*, N. B. McWilliams, Williamstown; *secretary*, Daniel N. Beers, Pittsfield; *treasurer*, Theodore W. Jones, Pittsfield; *executive councilor*, Helen M. Scoville, Pittsfield; *public relations councilor*, Patrick J. Sullivan, Dalton; *legislative councilor*, John Hughes, Pittsfield.

Bristol North — *President*, Joseph V. Chatigny, Taunton; *vice-president*, James H. Brewster, Attleboro; *secretary*, William E. Dawson, Taunton; *treasurer*, Charles E. Hoye, Taunton; *executive councilor*, Joseph L. Murphy, Taunton; *public relations councilor*, Milton E. Johnson, Attleboro; *legislative councilor*, William M. Stobbs, Attleboro.

Bristol South — *President*, Aubrey J. Pothier, New Bedford; *vice-president*, John S. Fielden, Fall River; *secretary and treasurer*, James E. Fell, Fall River; *executive councilor*, Richard B. Butler, Fall River; *public relations councilor*, Harold E. Perry, New Bedford; *legislative councilor*, Curtis C. Tripp, New Bedford.

Essex North — *President*, Robert E. Blais, Amesbury; *vice-president*, Z William Colson, Lawrence; *secretary*, Harold R. Kurth, Lawrence; *treasurer*, J. Leroy Wood, Lawrence; *librarian*, Frank W. Stockwell, Newburyport; *executive councilor*, Rolf C. Norris, Methuen; *public relations councilor*, Harold R. Kurth, Lawrence; *legislative councilor*, Nicandro F. DeCesare, Lawrence.

Essex South — *President*, Edward L. Peirson, Salem; *vice-president*, Burleigh B. Mansfield, Ipswich; *secretary*, Henry D. Stebbins, Salem; *treasurer*, Andrew Nichols, III, Danvers; *executive councilor*, Albert E. Parkhurst, Beverly; *public relations councilor*, Bernard Appel, Lynn; *legislative councilor*, Loring Grimes, Swampscott.

Franklin — *President*, Lawrence R. Dame, Greenfield; *vice-president*, Perrin N. Freeman, Greenfield; *secretary* and *treasurer*, Milton M. Sisson, Greenfield; *executive councilor*, Lawrence R. Dame, Greenfield; *public relations councilor*, John E. Moran, Greenfield; *legislative councilor*, Harold R. Mahar, Orange.

Hampden — *President*, Robert Hildreth, Westfield; *vice-president*, Arthur Riordan, Springfield; *secretary* and *treasurer*, Walter Williams, Springfield; *executive councilor*, Archibald Douglas, Westfield; *public relations councilor*, Frederic Hagler, Springfield; *legislative councilor*, Arthur Riordan, Springfield.

Hampshire — *President*, Edward J. Manwell, Northampton; *vice-president*, Henry Tadgell, Belchertown; *secretary* and *treasurer*, F. Mary P. Snook, Worthington; *librarian*, Abbie O'Keefe, Northampton; *executive councilor*, Maurice Kennedy, Hadley; *public relations councilor*, Joseph R. Hobbs, Williamsburg; *legislative councilor*, Justin E. Hayes, Northampton.

Middlesex East — *President*, Milton J. Quinn, Winchester; *vice-president*, Ira W. Richardson, Wakefield; *secretary*, Roy W. Layton, Melrose; *treasurer*, Charles W. DeWolf, Wakefield; *librarian*, Angelo L. Maietta, Winchester; *executive councilor*, Kenneth L. Maclachlan, Melrose; *public relations councilor*, Milton J. Quinn, Winchester; *legislative councilor*, Justin L. Anderson, Reading.

Middlesex North — *President*, James Y. Rodger, Lowell; *vice-president*, Harry Coburn, Lowell; *secretary*, Brendan D. Leahey, Lowell; *treasurer*, Mason D. Bryant, Lowell; *executive councilor*, William M. Collins, Lowell; *public relations councilor*, Samuel A. Dibbins, Lowell; *legislative councilor*, Joseph Sweeney, Lowell.

Middlesex South — *President*, John F. Casey, Allston; *vice-president*, James M. Baty, Belmont; *secretary*, Alexander A. Levi, Newton; *treasurer*, Fabyan Packard, Belmont; *orator*, Alfred O. Ludwig, Belmont; *executive councilor*, Joseph C. Merriam, Framingham; *public relations councilor*, Ralph H. Wells, Lexington; *legislative councilor*, Kenneth J. Tillotson, Belmont.

Norfolk — *President*, George W. Papen, Brookline; *vice-president*, W. Richard Ohler, Jamaica Plain; *secretary*, Basil E. Barton, West Roxbury; *treasurer*, Albert Ehrenfried, Brookline; *executive councilor*, Charles J. E. Kickham, Jamaica Plain; *public relations councilor*, Dean S. Luce, Canton; *legislative councilor*, Solomon L. Skvirsky, Brighton.

Norfolk South — *President*, Frederick Hinchliffe, Cohasset; *vice-president*, Edmund B. FitzGerald, Wollaston; *secretary* and *librarian*, Ebenezer K. Jenkins, South Braintree; *treasurer*, Raymond G. Vinal, Norwell; *executive councilor*, Robert L. Cook, Quincy; *public relations councilor*, Henry A. Robinson, Hingham; *legislative councilor*, David L. Belding, Hingham.

Plymouth — *President*, George A. Buckley, Brockton; *vice-president*, Mildred L. Ryan, Brockton; *secretary*, Samuel Gale, Brockton; *treasurer*, Henry Rabnowitz, Brockton; *librarian*, J. H. Weller, Bridgewater; *executive councilor*, George A. Moore, Brockton; *public relations councilor*, Charles D. McCann, Brockton; *legislative councilor*, Alfred L. Duncombe, Brockton.

Suffolk — *President*, Alexander J. A. Campbell, Brighton; *vice-president*, Lawrence J. McCarthy, Chelsea; *secretary*, Charles G. Shedd, Wellesley; *treasurer*, Sidney C. Wiggin, Waban; *executive councilor*, Harvey A. Kelly, Winthrop; *public relations councilor*, Howard F. Root, Boston; *legislative councilor*, William E. Browne, Boston.

Worcester — *President*, John J. Dumphy, Worcester; *vice-president*, Bancroft C. Wheeler, Worcester; *secretary*, Donald Hight, Worcester; *treasurer*, Arthur D. Ward, Worcester; *librarian*, Philip H. Cook, Worcester; *executive councilor*, John Fallon, Worcester; *public relations councilor*, Nicholas S. Scarcello, Worcester; *legislative councilor*, John B. Butts, Worcester.

Worcester North — *President*, Donald B. Cheetham, Athol; *vice-president*, C. Bertram Gay, Fitchburg; *secretary*, James G. Simmons, Fitchburg; *treasurer*, Frederick H. Thompson, Jr., Fitchburg; *executive councilor*, John J. Curley, Leominster; *public relations councilor*, James V. McHugh, Leominster; *legislative councilor*, C. Bertram Gay, Fitchburg.

Admissions Recorded from May 22, 1947, to May 22, 1948

YEAR OF ADMISSION	NAME AND RESIDENCE	MEDICAL SCHOOL
1947	Abbott, Richard N., Natick	Yale
1947	Abdu, Louis, Brockton	Tufts
1947	Abrams, Arthur L., Brookline	Harvard
1948	Alexander, Daniel David, E. Gardner	Middlesex
1948	Altenhaus, George, Rutland	University of Vienna
1948	Anderson, Ernest G., Belmont	Friedrich Wilhelm University, Berlin
1947	Appel, Charles F., Cambridge	Jefferson Medical College
1947	Armstrong, Beverly, Brighton	Syracuse University
1948	Armstrong, Catherine, Wellesley	University of Vermont
1948	Arnot, Robert E., Brookline	Harvard
1947	Avola, Francis A., Medford	Tufts
1948	Bacastow, Merle S., Worcester	University of Pennsylvania
1947	Ballantine, Henry T., Jr., Brookline	Johns Hopkins University
1947	Barger, A. Clifford, Brighton	Harvard
1948	Barkan, Donald B., Lynn	Boston University
1947	Barnes, John D., New Bedford	University of Maryland
1948	Barnes, William E., Taunton	Tufts
1948	Barnes, William L., Lowell	Middlesex
1947	Barone, Henry, Newton	Middlesex
1947	Barry, John J., Chelmsford	Middlesex
1947	Beer, Ernest G., Lowell	University of Heidelberg
1947	Belinkoff, Stanton, New Bedford	Middlesex
1948	Bell, William R., Marblehead	Yale
1948	Belock, John E., Beverly	University of Vermont
1947	Bergan, Carl A., Lenox	Middlesex
1948	Berman, Samuel S., Roxbury	Middlesex
1947	Bernstein, Joseph I., Springfield	Middlesex
1948	Bill, Alexander H., Jr., Boston	Harvard
1948	Bilski, Theodore D., Westfield	University of Halle, Germany
1948	Blacklow, Daniel J., Cambridge	Tufts

1948	Blaney, Lamson, Springfield	Cornell University
1947	Bloom, Bernard, Springfield	New York University
1948	Blotner, Carl, Lawrence	St. Louis University
1948	Blotnick, William, Medford	Middlesex
1947	Blumenthal, Frederick F., Bridgewater	University of Berlin
1947	Blumenthal, Irving J., Bedford	University of Freiburg
1948	Bonzey, Charles Merrill, Jr., Natick	Boston University
1948	Bradford, Martin L., Sharon	Boston University
1948	Bragg, Ernest A., Jr., Wellesley Hills	Boston University
1948	Brazelton, Thomas B., Boston	Columbia University
1947	Brean, Henry P., Boston	Harvard
1947	Breitwieser, Edna Ruth, Wellesley	University of Pennsylvania
1948	Briggs, Edward F., Truro	Baltimore Medical College
1947	Britton, Lee, Stoughton	Boston University
1948	Broderick, Thomas F., Jr., Boston	Tufts
1948	Bronson, Benjamin, Westfield	Middlesex
1948	Brooks, Oscar D., Dorchester	Middlesex
1948	Brown, Charles B., Rockport	University of Toronto
1947	Brown, Wesley C., Petersham	College of Physicians and Surgeons, Boston
1947	Brunner-Orne, Martha, Westwood	University of Vienna
1947	Burbank, Charles B., Brookline	Harvard
1947	Burke, John A., Holyoke	Tufts
1948	Burkhardt, Henry, So. Hadley	Wayne University
1948	Callahan, Charles L., Indian Orchard	Georgetown University
1947	Cannon, Raymond G., So. Boston	Middlesex
1948	Carey, Wm. A., Quincy	Yale
1947	Carp, J. Stanley, Saugus	Middlesex
1948	Carter, Max G., Boston	Harvard
1947	Cavanaugh, James E., Northampton	Columbia University
1947	Chapman, Carleton B., Boston	Harvard
1947	Clancy, Daniel H., Jr., Weymouth	Tufts
1947	Clark, John Q., Haverhill	Middlesex
1948	Clifford, William P. J., Worcester	Boston University
1947	Cohen, Milton, Brookline	Tufts
1947	Colby, Raymond G., Pittsfield	New York Medical College
1947	Coleman, Nathan W., Milton	Middlesex
1948	Collings, Robert Z., Jr., Fitchburg	University of Chicago
1948	Commons, Robert R., Framingham	Harvard
1947	Condon, Robert V., Roslindale	Tufts
1947	Conway, Raymond F., Holyoke	Boston University
1948	Cooper, Maurice Z., Wollaston	Long Island College of Medicine
1948	Coren, Simon, Attleboro	Middlesex
1948	Corwin, Harry J., East Longmeadow	Middlesex
1948	Costin, Maurice E., Framingham	Harvard
1948	Costrino, John J., Fall River	Tufts
1947	Crismond, Louis A., Quincy	George Washington University
1947	Crocker, Oscar, Pepperell	Middlesex
1948	Cronin, Thomas P., Jr., Somerville	Columbia University
1948	Crowell, David P., Wayland	Boston University
1947	Curtis, Ever, Gloucester	Tulane University
1948	Crudim, Myrtle B., Baldwinsville	Marquette University
1947	Dahl, Lewis K., Boston	University of Pennsylvania
1948	Davis, Stilman G., Jr., Salem	University of Vermont
1947	Del Campo, Dante A., Lynn	Boston University
1948	DelColliano, Michael R., So. Boston	Middlesex
1947	Dennen, William L., Wellesley	University of Rochester
1948	DeWeese, Byrne, Pittsfield	Kansas City University
1947	Dexter, Chester J., Brighton	Harvard
1947	Diamond, Israel, Boston	School of Medicine of Royal College of Edinburgh
1948	Dickson, William A., Jamaica Plain	Cornell University
1947	Dietel, Robert C., So. Hadley Falls	Jefferson Medical College
1948	Dixon, Stanley R., Springfield	University of Missouri
1947	Dodd, Wilson F., Mount Hermon	Cornell University
1947	Domenici, Trento J., Medford	Marquette University
1947	Doney, Frank C., Pittsfield	Middlesex
1947	Donnelly, James F., Rockland	Middlesex
1947	Donovan, Robert E., Arlington	Middlesex
1948	Dorfman, William A., Springfield	Middlesex
1947	Dowd, George C., Worcester	Middlesex
1947	Downing, William M., Boston	Boston University
1947	Drake, Emerson H., Dedham	Columbia University
1947	Drake, William L., Jr., Brookline	Marquette University
1947	Dubin, Max, Beverly	University of Vermont
1947	Dudis, Roger G., Athol	Albany Medical College
1947	Dunn, Paul P., Fall River	Tufts
1948	Dykens, James W., Worcester	University of Vermont
1947	Egan, Donald F., Springfield	Tufts
1947	Elgosin, Emid D., Millers Falls	McGill University
1947	Emerson, Charles P., Jr., Wellesley Hills	Harvard
1947	Ettenberg, Max	Middlesex
1947	Failla, John A., Greenfield	Boston University
1947	Fletcher, Mary E., Worcester	Syracuse University

1947	Flynn, John G., Swampscott	Boston University
1947	Foley, Joseph M., Dorchester	Harvard
1948	Fowler, Richard L., Spencer	University of Maryland
1947	Gates, Philip H., Cambridge	University of Minnesota
1948	Gephart, Francis T., Boston	Harvard
1948	Gewin, Edwin E., Gloucester	University of Tennessee
1948	Gilligan, Thomas J., Jr., Pittsfield	Columbia University
1947	Gleason, Charles S., Wareham	Tufts
1948	Glodt, Herbert R., Pittsfield	Tufts
1947	Goldstein, Robert, Brookline	Harvard
1947	Golub, Leon M., Dorchester	Boston University
1948	Goodwin, Arthur H., Wilbraham	Tufts
1947	Gorfine, Morris, Cambridge	Middlesex
1948	Gottler, Jacob, Dorchester	Boston University
1947	Grainger, Henry B., Tewksbury	Tufts
1948	Gray, Basil, Wellesley Hills	Syracuse University
1947	Gray, William, Brookline	University of Nebraska
1948	Green, Ross W., Worcester	Tufts
1947	Greenfield, Theodore B., Greenfield	Boston University
1948	Greer, William E. R., Jamaica Plain	Boston University
1947	Gregory, Irving F., Brighton	Tufts
1947	Grimes, Arthur J., Jr., Ipswich	Georgetown University
1948	Grogan, Richard H., Watertown	Harvard
1947	Guenther, Richard A., Medford	Tufts
1947	Haddad, Albert, Worcester	Albany Medical College
1948	Haentzschel, Lester E., Longmeadow	Washington (St. Louis University)
1948	Halpern, William, Brookline	University of Pennsylvania
1947	Halton, Edward J., Holyoke	Jefferson Medical College
1948	Halton, Gerald J., Holyoke	Cornell University
1948	Hamblin, William N., Westfield	Syracuse University
1947	Harrigan, Francis J., Salem	Middlesex
1948	Harris, Sidney, Worcester	New York University
1947	Hayek, Augusta, Brookline	University of Vienna
1947	Healy, Thomas, Worcester	Harvard
1947	Hebert, Roger D., Chicopee	Laval University, Quebec
1947	Hechler, Robert, Rutland Heights	Columbia University
1947	Helsel, Milton J., Lynn	Kansas City Medical College
1948	Hertzmark, Frederic, Dorchester	University of Buffalo
1947	Hester, James W., Cambridge	Tufts
1947	Hirschman, Kurt L., Pittsfield	University of Vienna
1948	Hochstadt, Otto, Brookline	University of Vienna
1948	Hoffman, Sumner H., Boston	Tufts
1948	Holden, Robert B., Boston	Harvard
1947	Homans, John, Jr., Brookline	Harvard
1947	Honig, Jerome L., Pittsfield	Duke University
1947	Horgan, John D., Cummington	New York Medical College
1948	Hubbell, John P., Jr., Brookline	Harvard
1948	Hunter, Henry J., So. Weymouth	Temple University
1947	Huntress, William W., Boston	Tufts
1948	Hurley, Francis E., Springfield	Tufts
1948	Hurley, Joseph P., Worcester	Tufts
1948	Hurley, Paul D., Jamaica Plain	Tufts
1948	Ingersoll, Robert E., Belmont	University of Rochester
1947	Inserra, Frank, Medford	Tufts
1948	Jacobs, Leon G., Quincy	Middlesex
1948	Jenney, William L., New Bedford	Yale
1948	Jennison, David B., Northampton	Harvard
1947	Johnson, Kingsland E., New Bedford	Middlesex
1947	Joseph, Alfred T., Cambridge	Tufts
1947	Jordan, Frank J., Jr., Springfield	Tufts
1948	Kalika, Karl, Lowell	Midwest Medical College
1948	Kaplan, Louis S., Salisbury	Middlesex
1948	Karlin, Harold, Brookline	Boston University
1947	Karras, Joseph D., Brookline	Middlesex
1947	Kaufman, Irving, Taunton	George Washington University
1947	Kaufman, Irving C., Auburndale	Long Island Medical College
1947	Kaye, Abraham, Worcester	Boston University
1947	Kefferstan, Frank J., Andover	Columbia University
1947	Keitlen, Philip B., Springfield	Eclectic Medical College, Cincinnati
1948	Kelly, John F., Hyde Park	Boston University
1947	Kent, Charles, Allston	University of Prague
1947	Kiarsis, Victor, New Bedford	Dartmouth
1948	Kiehl, Anna K., Boston	University of Kansas
1947	Koenig, Arthur T., Roslindale	New York Medical College
1947	Kroger, Louis E., Brighton	University of Cincinnati
1947	Krush, Thaddeus P., Newton	Western Reserve University
1947	Kubin, Ludwig, Waltham	University of Vienna
1947	Kurland, Leonard T., Brighton	University of Maryland
1947	Lane, Helen E., Boston	Boston University
1947	Larkin, John J., W. Roxbury	Harvard
1947	Lawrence, Bradford W., Attleboro	Tufts
1947	Lazansky, Joseph P., Belmont	University of Prague

1948	Leard, Samuel E., Boston	Boston University
1948	Leary, Gerald C., Westwood	Tufts
1947	Leavell, Hugh R., Cambridge	Harvard
1948	Lee, Charles F., Andover	Middlesex
1947	Levine, Sidney A., Melrose	George Washington University
1947	Lewis, Emery C., New Bedford	University of Vermont
1947	Licht, Sidney, Cambridge	New York University
1948	Lichty, Joseph S., Brookline	Harvard
1947	Lief, Philip A., Roxbury	New York University
1947	Lingos, John W., Brockton	Middlesex
1948	Lockshin, Abraham D., Medford	Middlesex
1948	Loeb, Elsie K., No. Attleboro	University of Munich
1947	Lucacer, Michael, Cambridge	Royal University, Palermo, Italy
1947	Lynch, J. P., Jr., Marlboro	Boston University
1947	MacKenzie, Michael V., Medford	Tufts
1947	Madoff, Irving, Clinton	New York University
1948	Maffeo, Peter A. R., E. Boston	Tufts
1947	Maison, George L., Needham	Northwestern University
1948	Malone, Edward H., Waban	Boston University
1947	Manning, John J., Worcester	University of Pennsylvania
1948	Martens, Theodore G., Fall River	University of Rochester
1948	Martin, Francis, Boston	Queens University, Canada
1947	Massik, Paul, Dorchester	Tufts
1948	Maynard, Guy B., Jr., New Bedford	Cornell University
1947	Mayo, Frederic B., Swampscott	Cornell University
1948	Mayo, Walter V., Kingston	Middlesex
1948	McCarter, Robert H., Boston	Jefferson Medical College
1948	McCarthy, Francis D., Melrose	Tufts
1948	McDermott, John R., Boston	Yale
1947	McEneaney, Joseph P., Lawrence	Boston University
1947	McEnroe, Donald F., Whitman	Middlesex
1947	McGinty, John F., Jr., Lawrence	Jefferson Medical College
1947	McKernan, Bernard F., Upton	Middlesex
1947	McLean, Donald E., Winchester	Harvard
1947	McMahon, Francis J., Holbrook	Boston University
1948	McManama, John C., Waltham	Tufts
1948	McVay, John F., Dedham	Tufts
1947	Meilman, Edward, Boston	Harvard
1947	Meislin, Jacob, Bedford	University of Lausanne
1947	Meline, David I., West Roxbury	Middlesex
1948	Merlis, Jerome K., Framingham	University of Louisville
1947	Meshorer, Edward, Quincy	Kansas City University of Physicians and Surgeons
1948	Michaelson, Abraham I., Lynn	Middlesex
1948	Michaud, Raymond R. A., Salem	Tufts
1947	Mickiewicz, Matthew S., Northampton	New York University
1947	Milden, Forrest, Haverhill	Middlesex
1947	Millen, Hyman, Dorchester	Middlesex
1948	Miller, Nathaniel B., Haverhill	Middlesex
1948	Mills, Waldo O., Brookline	University of Oregon
1948	Moll, Frederic C., Boston	University of Rochester
1948	Moll, Gretchen H., Boston	Yale
1947	Monahan, John L., Dorchester	Tufts
1948	Moore, Frederick T., West Springfield	Tufts
1947	Moore, Robert L., Jr., Worcester	Boston University
1947	Morris, John M., Boston	Harvard
1948	Morrison, Jonathan I., Brookline	Dalhousie University
1947	Mulligan, William J., Brookline	University of Vermont
1947	Murphy, Rosemary A., Boston	Boston University
1948	Murray, Edward S., Boston	University of Iowa
1947	Nalband, Edward H., Wollaston	Tufts
1947	Nelson, Robert E., Arlington	University of Oregon
1948	Nelson, Rosemary, Boston	University of Chicago
1948	Neves, Edmund F., Fall River	Jefferson Medical College
1947	Newcombe, Richard V.	University of Vermont
1947	Newerla, Gerhard J., Waltham	Middlesex
1948	Newman, Walter, Springfield	University of Graz (Austria)
1947	O'Hare, James M., Chestnut Hill	Harvard
1947	Orbach, Rudi J., Quincy	University of Berlin
1948	Orr, Robert B., Boston	University of Virginia
1947	Orris, Israel, Lynn	Wayne University
1947	Pagel, Max M., Newton Centre	University of Breslau
1948	Pagliuca, Gerald F., Medford	College of Physicians and Surgeons, Boston
1947	Paster, Harold I., Brockton	Middlesex
1947	Paul, Oglesby, Boston	Harvard
1947	Peloquin, Laval U., Lowell	Georgetown University
1947	Pendola, Joseph J., Longmeadow	Hahnemann Medical College
1948	Pereira, Manuel C., Fall River	Lyceum of Ponta Delgada, Azores
1947	Perkins, Herbert A., Lynn	Tufts
1947	Perry, Helen N., Boston	Tufts
1948	Peters, James M., Newton Centre	Tufts
1947	Petrell, N. R., Plymouth	Middlesex
1947	Picariello, Americo, Somerville	Middlesex

1947	Pick, Walter, Boston	Harvard
1947	Pious, Lawrence K., Lexington	Middlesex
1947	Polatin, Sydney, Roslindale	Middlesex
1947	Potsubay, Samuel F., Holyoke	Harvard
1948	Powers, Harry T., Fall River	Cornell University
1948	Putnam, Hazel E., Plymouth	McGill University
1947	Quinn, Edmund P., Medford	Tufts
1947	Racioppi, Francesca M., Wellesley	Boston University
1948	Ramsay, Beatty H., Brookline	University of Manitoba
1947	Rand, Harry, Dorchester	Middlesex
1947	Raphael, Sumner I., Brighton	Tufts
1948	Rappaport, David, Oak Bluffs	Middlesex
1947	Reagan, Daniel J., Worcester	Harvard
1947	Reid, David H., Melrose	Tufts
1948	Reppucci, Anthony, Lowell	Middlesex
1947	Richmond, Paul, Jr., Worcester	Georgetown University
1948	Riemer, Karl, Boston	Tufts
1947	Riemer, Robert W., Norwood	Tufts
1947	Riggs, Benjamin C., Topsfield	Columbia University
1947	Riley, Leo H., Jr., Newton	Boston University
1948	Risley, Thomas S., Boston	Harvard
1948	Robertson, Charles W., Boston	Syracuse University
1948	Rockwood, Lawrence, Medford	Tufts
1947	Rogers, Joseph, Boston	Harvard
1947	Roopenian, Aram, South Boston	Tufts
1947	Rosen, Daniel, Roxbury	University of Vienna
1947	Rosenbaum, Jack D., Framingham	Yale
1947	Rosenberg, Isadore N., Boston	Harvard
1948	Rosengard, David E., South Boston	Tufts
1948	Ross, Helen M., Boston	Tufts
1948	Rowbotham, John L., Canton	Harvard
1947	Rowell, Russell J., Beverly	Tufts
1947	Roy, James E., Worcester	Harvard
1947	Sands, Herman C., Salem	Middlesex
1947	Sapareto, Rocco L., Haverhill	University of Maryland
1947	Sapienza, Peter L., Everett	Boston University
1947	Scannell, John G., Belmont	Harvard
1948	Schlessinger, Paul J., Cambridge	Harvard
1947	Schultz, John D., Jr., Boston	Johns Hopkins University
1948	Scoville, Peter G., Hyde Park	College of Physicians and Surgeons, Boston
1948	Sears, Robert A., Brookline	Yale
1948	Seeler, Albert O., Boston	Harvard
1946	Selverstone, Bertram, Belmont	Harvard
1947	Senesac, Archibald E., New Bedford	Tufts
1947	Sennott, Francis W., Cambridge	Tufts
1947	Shauffer, Irving A., Brookline	Tufts
1947	Shaw, John E., Newburyport	Georgetown University
1948	Shea, Daniel J., Jr., Beverly	Boston University
1948	Shuman, Samuel B., Dorchester	Middlesex
1948	Siegel, Henry W., Dorchester	Middlesex
1947	Silver, Joseph M., Fitchburg	Middlesex
1948	Simon, Aaron I., Fitchburg	Ohio State University
1948	Skornik, Nathan, W. Newton	Middlesex
1947	Snyder, Franklin F., Newton Centre	Johns Hopkins University
1948	Sotirion, George A., Springfield	Duke University
1947	Soule, Robert M., Melrose	Harvard
1947	Souliotis, Paul T., Cambridge	Hahnemann Medical College
1947	Sparling, Harold J., Whalom	Boston University
1947	Sparling, Seda A., Whalom	Boston University
1947	Spelfogel, Benjamin, Mattapan	Middlesex
1947	Spelman, Philip J., Plymouth	Columbia University
1947	Spindler, James F., Boston	University of Georgia
1947	Spitzer, Maurice G., North Adams	Middlesex
1948	Stewart, Goodwill M., Brockton	Tufts
1947	Stoddard, Eben, Salem	Rush Medical College
1948	Stone, Bartlett H., Brookline	University of Vermont
1947	Stone, Timothy P., Southboro	Tufts
1947	Straccia, Frank A., Hawthorne	Tufts
1947	Streeter, C. Clarke, Harvard	Tufts
1948	Sullivan, Thomas H., Fall River	Middlesex
1947	Sweet, William H., Brookline	Harvard
1947	Szydlowski, Sigmund J., Wakefield	Middlesex
1948	Taft, Edgar B., Cambridge	Yale
1948	Taggart, William G., Westfield	Jefferson Medical College
1948	Talbot, Timothy R., Jr., Boston	University of Pennsylvania
1948	Tannenbaum, Harold S., Jamaica Plain	Middlesex
1947	Tatlock, Hugh, Northampton	Harvard
1948	Tauber, Joseph, Springfield	University of Vienna
1947	Tedeschi, Cesare G., Framingham	University of Bologna
1948	Terranova, Anthony J., Attleboro	Middlesex
1948	Thatcher, Lyndon H., Dennis	Columbia University
1947	Thompson, John R., Dalton	McGill University

1947	Thompson, William J., Canton	Middlesex
1947	Timberlake, William H., Cambridge	Tufts
1947	Tolpin, Isidor, Lynn	Middlesex
1947	Trakas, John C., Milton	Harvard
1947	Trott, Arthur W., Wollaston	Harvard
1947	Troubalos, Stephen, Brighton	Marquette University
1947	Tye, Mauray J., Haverhill	Tufts
1948	Valenstein, Arthur F., Boston	Cornell University
1948	VanKeuren, Howard C., Weymouth	Albany Medical College
1947	Vaughan, Robert H., Dedham	Harvard
1948	Velon, Bruce P., Taunton	Middlesex
1947	Visconti, James J., West Springfield	Midwest Medical College
1947	Waldinger, Clara, Jamaica Plain	University of Vienna
1948	Wallace, Robert W., Westwood	University of Pittsburgh
1948	Waring, George W., Jr., Brookline	Johns Hopkins University
1947	Warner, Frank A., Boston	Harvard
1947	Webster, Earle H., Hyannis	Harvard
1947	Weinreb, Joseph, Newton Centre	Vanderbilt University
1947	Weinstein, Max I., Lawrence	Middlesex
1948	Weiss, Daniel, Dorchester	Boston University
1947	Weiss, Samuel, Brockton	Tufts
1948	Weller, Thomas H., Boston	Harvard
1947	West, George V., Reading	Tufts
1947	Whiting, George L., Abington	Tufts
1947	Whitney, Byron V., Bangor, Maine	Tufts
1948	Wholey, John J., Lawrence	Middlesex
1947	Whorton, Carl M., Squantum	Vanderbilt University
1948	Wilder, Francis D., Brockton	University of Lausanne
1948	Wilker, Sydney R., Roxbury	Boston University
1947	Wilson, Norman J., Boston	University of Maryland
1947	Wilson, Norman L., Quincy	University of Virginia
1947	Winsten, Sol Stephen, Northampton	University of Berne
1948	Witmer, Donald S., Fall River	College of Physicians and Surgeons, Boston
1947	Wixted, Francis J., East Milton	New York Medical College
1948	Woodman, Ernest L., Jr., Lawrence	Tufts
1948	Woodruff, Charlotte S., Pittsfield	Long Island College of Medicine
1947	Wool, Jack, Boston	University of Vermont
1947	Yaukopolus, Konstantine G., New Bedford	Boston University
1947	Yatsuhashi, Masao, Brookline	Harvard
1947	Yosko, Norman, E. Longmeadow	Chicago Medical School
1947	Yudell, Milton H., Worcester	Rush Medical College
1947	Zamcheck, Norman, Boston	Harvard
1947	Zanconato, Guerrino J., West Stockbridge	Tufts
1947	Zarecki, Peter S., Jr., Lynn	Tufts
1948	Zawislak, Joseph J., Lawrence	Middlesex
1947	Zielinski, Joseph F., Holyoke	Jefferson Medical College

Deaths Reported from May 22, 1947 to May 22, 1948

ADMITTED	NAME	PLACE OF DEATH	DATE OF DEATH	AGE
1934	Louis Alpert	Middleboro	August 17, 1947	44
1906	Louis Arkin	Sharon	October 1, 1947	70
1917	Avedis D. Avedisyan	Brighton	November 16, 1947	75
1894	Silas H. Ayer	Boston	September, 1947	86
1906	John B. Bain	Boston	October 13, 1947	68
1900	†John A. Barton	Fitchburg	1948	74
1912	Ewald G. Baum	Canaan, New Hampshire	October 22, 1947	63
1925	G. R. Baxter	Boston	July 24, 1947	52
1912	Charles F. K. Bean	West Medford	September, 1947	60
1904	Orrin C. Blair	Lynn	August 3, 1947	68
1879	Walter P. Bowers	Clinton	July 22, 1947	92
1930	Melville G. Boyd	Ontario	September, 1947	46
1907	James C. Bright	Fall River	November 12, 1947	74
1893	Martin M. Brown	North Adams	September 19, 1947	84
1942	William G. Cole	Bermuda	April 16, 1948	46
1910 1919	George J. Connor	Haverhill	April 1948	62
1909	Charles L. Curtis	Salem	December 11, 1947	62
1916	Elliott C. Cutler	Brookline	August 16, 1947	59
1895	†Charles B. Darling	Waban	April 19, 1948	79
1905	Hilbert F. Day	Cambridge	May 17, 1947	68
1885	Samuel Delano	New Britain, Connecticut	November 18, 1947	89
1896	Isaac S. F. Dodd	Pittsfield	January, 1948	80
1908	Arthur H. Ellis	Greenfield	March 14, 1948	66
1880	†Frederick W. Ellis	Newton Centre	April 30, 1948	90
1905	†Frederick L. Everett	Watertown, New York	March 6, 1948	76
1897	John E. Fish	Canton	March 30, 1948	75
1902	†Elisha Flagg	New York	June 8, 1947	82
1914	Edwin R. Fleming	Medford	March 4, 1948	69

1920	Edward H. Ganley	Methuen	January 23, 1948	57
1919	Walter T. Garfield	Belmont	May 31, 1947	65
1947	Maurice A. Gilbert	Chelsea	January 18, 1948	61
1909	Florence Gilman	New Hampshire	August 1, 1947	69
1925	Edwin B. Goodall	Boston	August 6, 1947	65
1905 / 1910	George H. Gorham	Jamaica Plain	March 12, 1948	76
1932	Henry M. Grady	Dedham	July 30, 1947	—
1905	Arthur M. Greenwood	Marlboro	December 14, 1947	71
1908	Henry Hawkins	Sullivan, Maine	September 4, 1947	74
1899	Thomas R. Healy	Newburyport	February 25, 1948	67
1911	Francis F. Henderson	Milton	November 20, 1947	62
1912	Thomas F. Hennessey	Swampscott	July 21, 1947	60
1914	George V. Higgins	Randolph	September 9, 1947	67
1913	Samuel Hoberman	Malden	February 18, 1948	66
1906	†Walter A. Hosley	Topsfield	March 25, 1948	70
1885	†Oliver H. Howe	Cohasset	March 11, 1948	87
1917	Winfred L. Howe	Everett	August 18, 1947	72
1903	Ernest L. Hunt	Worcester	January 17, 1948	70
1926	†Reid Hunt	Boston	March 10, 1948	78
1921	Vincent J. Irwin	Springfield	February 26, 1948	60
1894	Mary W. L. Johnson	Brighton	September 15, 1947	80
1903	Raymond C. Jones	Ashby	December 2, 1947	67
1914	Ernest H. Judd	Springfield	October 17, 1947	62
1880	Patrick H. Keefe	Providence, Rhode Island	October, 1947	93
1931	Connie H. King	Duxbury	January 22, 1948	41
1902	Frederick A. King	Marshfield	September 14, 1947	—
1910	George C. King	Fall River	December 21, 1947	61
1901	Frederick D. Lambert	Tyngsboro	April 20, 1948	70
1919	Leslie P. Leland	Worcester	March 16, 1948	63
1947	William J. Lord	Great Barrington	December 14, 1947	40
1927	Henry Lubinsky	Fall River	May 25, 1948	45
1897	†James A. Mansfield	Dorchester	June 29, 1947	81
1916	†James V. May	Watertown	December 24, 1947	76
1907 / 1942	†Leo T. McCready	Jamaica Plain	May 5, 1948	68
1882	†Herbert McIntire	Cambridge	April 21, 1948	91
1923	Archibald McMillan	Athol	January 16, 1948	76
1883	†William H. McOwen	New York	March 9, 1948	75
1920	Leon J. Menard	Fall River	August 23, 1947	55
1928	†Grace Milliken	Brookline	January 19, 1948	75
1930	Herman A. Morrill	Gardner	May 28, 1947	49
1917	Harold A. Murphy	Jamaica Plain	September 23, 1947	55
1900	James Nightingale	Worcester	July, 1947	72
1921	Robert N. Nye	Chestnut Hill	September 10, 1947	55
1912	E. J. O'Brien, Jr.	Boston	February 16, 1948	61
1917	†John C. O'Brien	Turners Falls	March 18, 1948	84
1932	Carver H. Osborne	Brookline	January 3, 1948	51
1920	William J. Pelletier	Turners Falls	November 16, 1947	50
1940	Anthony S. Pendola	Holyoke	January 10, 1948	58
1897	†Ernest D. Pillsbury	Stoughton	January 6, 1948	79
1932	Mendel Poliak	Springfield	September 15, 1947	60
1910	John A. Reese	Attleboro	December 10, 1947	62
1922	Frederick Reis	Jamaica Plain	February 4, 1948	72
1943	John D. Rice	West Boylston	October 4, 1947	32
1918	Carroll H. Ricker	Worcester	September 6, 1947	79
1945	Albert I. Robbins	New Haven, Connecticut	April 14, 1948	34
1936	Leon J. Robinson	Springfield	February, 1948	42
1915	Moses V. Safford	Jamaica Plain	June 20, 1947	80
1891	James A. Shatswell	Beverly	August 26, 1947	72
1908	Benjamin E. Sibley	Brookline	November 19, 1947	73
1900	Albert E. Small	Boston	June 9, 1947	72
1894	Frederick G. Smith	Somerville	January 9, 1948	81
1912	Elizabeth C. Underhill	New York	September 21, 1947	76
1922	John M. Wise	Watertown	August 7, 1947	71

†Retired fellow

Total number of deaths of active fellows	76
Total number of deaths of retired fellows	15
Grand total	91

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

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CASE 34311

PRESENTATION OF CASE

A seventy-five-year-old Negro was admitted to the hospital complaining of pain in the right flank.

About six years before admission the patient began having superficial pain in the right lower thigh. No other accompanying signs or symptoms were noted. Four months before admission there was sudden weakness of the right leg, and standing caused severe pain. Since then he had been unable to walk. There was no joint pain, chills or fever. About two months before admission there was occasional mild right-flank pain, which did not bother the patient unduly. Defecation and urination were normal at that time, and there were no gastrointestinal symptoms. The pain later became severe and persistent and occasionally radiated to the right groin. The patient's wife stated that he had had black stools. She also stated that he had been under treatment for "high blood pressure" for four years and that eight weeks before admission he had an episode of very labored breathing. He had had an easily reducible right scrotal hernia for fifty years.

Physical examination revealed a poorly nourished, confused, dyspneic and acutely ill man. The veins of the scalp and neck were engorged but not pulsating. The lungs were emphysematous. Medium moist rales were heard throughout both bases. The right leaf of the diaphragm was much elevated and showed limited excursion. The heart was enlarged to the left. A Grade II systolic murmur was heard over the whole precordium. There was moderate distention of the abdomen, and peristalsis was normally active. Voluntary resistance was encountered over the right flank, which was filled by a tender, poorly defined mass. Little, if any, costovertebral angle tenderness was elicited. Rectal examination revealed tenderness on the right. The prostate was slightly enlarged and nodular. A large, right scrotal mass was present, in which gurgles were heard. There was superficial and deep tenderness on the anterior aspect of the right thigh. Knee jerks were normal. No edema was present.

The temperature was 97.5°F., the pulse 90, and the respirations 22. The blood pressure was 100 systolic, 68 diastolic.

Examination of the blood revealed a white-cell count of 23,000, with 88 per cent neutrophils, and a red-cell count of 4,000,000. The hemoglobin was 12 gm. The urine had a specific gravity of 1.019 and contained + albumin. No casts were seen, but the sediment was "loaded" with leukocytes. The stool was brown and had a ++ guaiac reaction. The nonprotein nitrogen was 42 mg., the serum calcium 7.7 mg. and the phosphorus 3.0 mg. per 100 cc. The alkaline phosphatase was 4.8 and the acid phosphatase 1.4 units per 100 cc. The total serum protein was 5.7 gm. per 100 cc. The chloride was 94 and the carbon dioxide 21.2 milliequiv. per liter.

X-ray examination of the chest showed an elevated diaphragm, linear streaks of atelectasis at the right base, enlargement of the heart, and elongation and tortuosity of the thoracic aorta. No free air was seen beneath the diaphragm. There was extensive Paget's disease involving the pelvis and lumbar spine. A diffuse increased density was seen in the right side of the abdomen. Neither kidney contour was visualized, and the right psoas shadow was not present. There was a single large calcification, about 1 cm. in diameter, and several smaller calcifications superimposed on the right kidney shadow. Calcification was seen in the abdominal aorta. An excretory urogram revealed an essentially normal left upper urinary system. No dye was excreted from the right kidney. A barium enema was negative except for the scrotal hernia, which contained cecum and small bowel. A retrograde pyelogram was attempted but the catheter would not pass up the ureter more than 0.5 cm. No efflux was seen from either orifice.

On the fourth hospital day an operation was performed.

Differential Diagnosis

Dr. Edward L. Young: A medical man must have made the statement about the mass in the right flank. If it had been made by a trained urologist, he would have said whether or not it gave the impression of going through to the flank on ballottement; if it is kidney, there is generally the sensation that the mass goes through or close to the costovertebral angle.

The blood picture is surprisingly good for a man as sick as this.

Let us discuss the x-ray films at this point.

Dr. Stanley M. Wyman: The heart is considerably enlarged; it looks to be chiefly left ventricular enlargement. The aorta is quite tortuous, especially in the descending portion, and there is some calcification in the wall. The ascending portion of the aorta appears dilated and may be dilated as far as the descending portion, although one cannot be sure of the latter. There is some atelectasis in the lower portion of the right lung field. I cannot localize it because there is no lateral view. The humerus shows some changes quite consistent with Paget's disease. The films of the abdomen show the area of calcification described, overlying the right kidney; in the lateral view it is seen to lie far posteriorly, again coincident with the kidney. I cannot outline the kidney itself on either side with any degree of certainty. The intravenous dye is excreted by the left kidney in fair concentration, and the major and minor calyxes are grossly normal so far as can be determined. The kidney appears approximately normal in size. There are several films that show a faint linear shadow in the region of the right kidney, which makes me wonder if it could be the medial margin of the kidney. If so, the kidney is displaced laterally. However, this is not certain enough to make any definite statement. There is a well defined arc of calcification just to the left of the lumbar spine, which in the lateral view is seen to be a considerably dilated abdominal aorta. The bones of the lumbar spine and pelvis are normal.

Dr. Young: Are you sure that what you see in the bones is Paget's disease and not metastases?

Dr. Wyman: I think it is Paget's disease.

Dr. Young: Are you always able to differentiate?

Dr. Wyman: No. The differential point is the increased size of the bone and the involvement chiefly of the cortex. These are fairly definite in this particular person. The thickening in the ilium lies along the margin and inner aspect of the pelvis. Paget's disease is more likely than osteoblastic metastases.

Dr. Young: Will you comment on the reason why you think there is stone in the kidney?

Dr. Wyman: The character of the calcification is rather discrete. It is not homogeneous but mottled. It lies far posteriorly, at the expected location of the kidney itself. All these add up to make it more likely to be a kidney stone than a calcified lymph node or a gallstone. I think the shadow in the right flank, which is described in the protocol, may represent the colon filled with fluid because on the barium enema there is a large loop of colon overlying the ilium, and one can see a big hernial sac containing cecum and ileum.

Dr. Young: The whole picture seems to me to be somewhat confusing. We have an old man who is sick. The pain in his leg may have been due to hypertrophic changes, which are usually seen in the spine in a man of seventy-five, or to Paget's disease. The black stools are difficult for me to put into the picture. That statement was made by his wife but apparently no attempt was made to find out whether medication had been given that might have caused them. We are not sure that they were due to blood. He was treated for high blood pressure four years ago — and yet he comes in with a blood pressure far below what is expected. We can attribute that to his poor general condition. He has a leukocytosis, a mass in the right flank, a shadow that suggests kidney stone, and pus in the urine. The right ureter could not be catheterized. That does not necessarily mean that it was completely blocked. It often is difficult to catheterize the ureter because of stiffness — when actually there is a passageway through it. The white count suggests that there was infection going on even though he had no temperature. The other kidney was working. The specific gravity was 1.019. The nonprotein nitrogen did not show much elevation; in fact, 42 mg. would be within high normal limits.

The first thought here would be a calculus pyonephrosis, but one must remember that although tuberculosis of the urinary tract almost always shows itself in bladder irritation, it occasionally occurs without it. It is not impossible that there is a background here of tuberculosis and mixed infection, although that is not usual. We must not forget that tumor of the kidney can have a stone present. There was a negative barium enema, and I cannot seem to connect the blood in the stool with this unless we have a condition of congestion due to cardiac decompensation. So I think that this constitutes a calculus pyonephrosis, with or without one of the other two things being present. He is an extraordinarily poor risk because of what we know about his general condition; yet today with the ability we have to increase the body chemistry of the patient to a level more closely normal we can get away with what ten or fifteen years ago would have been murder. If this is calculus pyonephrosis, a two-stage operation may give him a chance. There may be something entirely different, but I will put calculus pyonephrosis as my first choice, with or without the other.

DR. WYMAN: I do not believe that it is safe for us to say that the kidney on the right is displaced laterally. There is a faint hint from the available films that it is farther lateral than usual. The position of the stone is more lateral than one would see with pelvic stone.

DR. YOUNG: Is there a chance that this mass of which you speak and which I have disregarded could be an aneurysm?

DR. WYMAN: I was thinking of that; but I cannot outline calcium in it with any degree of certainty. There is a fair amount of calcium lying up here. I do not know what it is.

DR. YOUNG: The description does not suggest that the mass pulsated.

DR. WYMAN: I wondered about an aneurysm of the renal artery or some such thing pushing the kidney up.

DR. YOUNG: It did not pulsate.

CLINICAL DIAGNOSIS

Calculus pyonephrosis.

DR. YOUNG'S DIAGNOSIS

Calculus pyonephrosis.
Tumor of kidney?
Tuberculosis of kidney?

ANATOMICAL DIAGNOSES

Retroperitoneal fibrosarcoma, with pressure on right kidney, extension into inferior vena cava, duodenum, and ascending colon, and with metastases to lung.
Perinephritis, acute, chronic.
Peritonitis, acute.
Operation: nephrostomy, right.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: The kidney was explored through a right transverse incision and chronically inflamed perirenal tissue was encountered as the lumbodorsal fascia was opened. At that time aspiration of the tissue yielded thick, greenish pus. An incision was made through the kidney cortex into the pelvis and the stone, seen by x-ray, was removed. A nephrostomy tube was placed in the pelvis. At the time of operation a biopsy of some of this inflamed tissue around the kidney was taken on which a diagnosis of fibrosarcoma was made. The patient did not do well postoperatively and died a few days later.

At autopsy we found that the kidney was displaced a great deal; in fact, it lay almost horizontally. This horizontal position of the kidney was due to a large retroperitoneal mass in the right side of the abdomen which pushed the kidney up. Anterior to the mass was the ascending colon and medially and superiorly was the duodenum. They were both intimately connected by the mass, which had eroded through into their lumens. This adequately explains the tarry stools noticed by the patient's wife and the positive guaiac test in the hospital. The mass was tremendous in size and proved to be a fibrosarcoma arising in the retroperitoneal space. A very interesting finding was an extension of the tumor into the inferior vena cava in a polypoid fashion. Microscopic sections of the surface of this polyp showed endothelium of the vena cava, so that the tumor was really pushing the vena cava ahead. The tumor did not arise within the kidney substance at all, the fibrous capsule of the kidney being clearly visible between renal parenchyma and tumor. The tumor had metastasized to the lung.

DR. YOUNG: You did not check the bone?

DR. CASTLEMAN: One section of bone showed no evidence of metastases. The pus that the operator had found at operation was due to the perforation of the ascending colon with spillage of fecal material.

DR. YOUNG: Between the tumor and the ascending colon?

DR. CASTLEMAN: Yes. At the time of autopsy that had extended up along the right gutter into the peritoneal cavity and had produced peritonitis on the right side.

DR. YOUNG: Did they get through to the kidney?

DR. CASTLEMAN: No, there was no stone in the kidney.

DR. YOUNG: But they got into the pelvis?

DR. CASTLEMAN: Yes, the nephrostomy tube was in the kidney pelvis, but the tumor had completely obstructed the ureter on that side.

DR. YOUNG: Did the tumor involve the nerve to cause the leg symptoms?

DR. CASTLEMAN: I cannot answer that. It might well have.

CASE 34312

PRESENTATION OF CASE

A seventy-one-year-old man entered the hospital because of an abdominal mass.

The abdomen had always been of rather extensive proportions, but three months before admission he noted a hard swelling on the right side which did not change appreciably in size following discovery. His appetite was always good although for three years he was bothered by increasing belching following meals. There was never any food intolerance, abdominal pain or vomiting. His diet had always been good. Gradually increasing constipation also bothered him during the three years before admission, although there was no change in the character of the stools and never any diarrhea. There were never any symptoms of liver disease and no weight loss.

The patient was born in Greece, where he was brought up on a farm. He came to the United States at the age of thirty-one and went to work in a shoe

factory. He continued in this business until admission to the hospital and was exposed to numerous types of chemicals. This exposure occasioned visits to the skin clinic of the hospital twice several years previously at which time diagnoses of contact dermatitis were made.

Physical examination showed a well developed but poorly nourished man. Examination of the chest was not remarkable except for a high relatively fixed diaphragm. The abdomen was large and asymmetrical. There was a fluctuant, nontender, apparently lobulated mass, which moved with respiration and extended from the fourth intercostal space on the right to two inches below the umbilicus and could be felt in both flanks, being more prominent on the right. It could not be transilluminated. There were bilateral reducible inguinal hernias and bilateral varicoceles.

The temperature, pulse and respirations were within normal limits. The blood pressure was 148 systolic, 90 diastolic.

The blood, urine and stool tests were negative except for slight anemia, a hemoglobin of 11.5 gm., and a 10 per cent eosinophilia. The nonprotein nitrogen, fasting blood sugar, amylase, cephalin flocculation, van den Bergh, and bromsulfalein test, phosphorus, alkaline phosphatase, and prothrombin time were all within normal limits. The total protein was 8 gm. per 100 cc., with an albumin-globulin ratio of 0.85. An x-ray film of the chest showed nodularity of the right diaphragm. A gastrointestinal series showed a negative esophagus, a stomach displaced to the left and posteriorly, and no intrinsic involvement of the gastrointestinal tract. It was thought that the mass was related to the liver. There were a number of calcifications in the upper portion of the liver. An echinococcus skin test was positive.

On the sixth hospital day an operation was performed.

Differential Diagnosis

Dr. Alfred Kranes: There are two statements in the record to which I should like to take exception. One is the description of the mass, which is said to extend from the fourth intercostal space on the right to two inches below the umbilicus. I do not see how one can feel a mass of this size in the fourth intercostal space. What is meant is that the area of liver dullness extended up to the fourth intercostal space. I find it hard to believe that the mass could be felt that high, although that is the impression one gets from the description. One can only say that the mass extended so far below the costal margin. The other statement that I question is the description by x-ray of nodularity of the diaphragm. I think what is meant is irregularity. One cannot say that the diaphragm is nodular, can one?

Dr. Stanley M. Wyman: No.

Dr. Kranes: That implies a third dimension which, of course, we do not have.

Dr. Wyman: The radiologist's statement in the record was, "There is irregularity of the right leaf of the diaphragm resulting from nodularity or masses projecting from the superior aspect of the liver."

Dr. Kranes: That makes the diagnosis easier.

Dr. Wyman: The right leaf of the diaphragm is definitely elevated and as you see comes to the fourth interspace. The lung fields otherwise appear intrinsically clear. The heart shadow is prominent toward the left, probably in the region of the left ventricle. The barium-filled stomach is faintly seen in this film displaced toward the left, and the duodenal cap is displaced downward and to the left. The entire right side of the abdomen is dense and there is a suggestion of several small areas of calcification. There is a very definite mass, nodular and lobulated, on the right.

Dr. Kranes: As I see this problem there is no differential diagnosis. What else can this be but an echinococcus cyst? If that diagnosis is correct, and I can see no other one to make, I must say that I would not have him operated on. You must remember that he is seventy-one years old. The disease started before the age of thirty-one and although he gave a history of having felt the mass only three months before entry, you will all agree, I am sure, that an echinococcus cyst of this size must have been growing for many, many years. Furthermore, despite its size, and it does seem to be huge, it has produced absolutely no symptoms. The record states that the man was feeling quite well except for a few mild symptoms of belching and constipation. Nor has it interfered with the function of any organ although it has displaced the stomach. While it may involve a fairly large part of the liver, it has not interfered with the liver's function or produced any alteration in the laboratory tests. They were all normal. Consequently, I probably would not have advised this patient to be operated on since I see nothing to be gained by operation at seventy-one on an asymptomatic echinococcus cyst. The chances are that he might have gone another five or ten years still

asymptomatic with a fairly large mass in his abdomen.

Could we be wrong about the diagnosis? Unfortunately, yes. However, the mass is lobulated and apparently cystic — and I know of no other cystic mass that could grow to this size in the right upper quadrant without producing symptoms. Any solid mass of this size would undoubtedly produce symptoms. The mass was certainly cystic. Those who took care of him thought so because they describe it as fluctuant and went so far as to try to transilluminate it. If it did not involve the under surface of the right diaphragm one would have to consider such things as large multilocular pancreatic cysts — they grow to huge proportions. If it were on the left, a splenic cyst would have to be thought of, or an omental cyst. But under the circumstances there is nothing to indicate any other diagnosis but an echinococcus cyst.

How do you feel about it, Dr. Adams?

Dr. F. Dennette Adams: I agree that it would be a job to get it out and the chances for spilling are great.

Dr. Kranes: The risk of spilling seems greater than the risk of leaving it alone. He apparently did well.

Clinical Diagnosis

Echinococcus cyst.

Dr. Kranes's Diagnosis

Echinococcus cyst.

Anatomical Diagnosis

Echinococcus cyst.

Pathological Discussion

Dr. Ronald C. Sniffen: The patient was operated on with a preoperative diagnosis of echinococcus cyst, and it was found that the cyst ran from the pelvis up to the liver and occupied almost the entire right lobe of that organ. It was surrounded by peritoneal adhesions, and daughter cysts were noticed in the remaining liver. A partial excision of the cyst was performed and a drain was inserted into the remaining cavity. Microscopically the wall of the cyst showed the characteristic laminated chitinous membrane and germinative epithelium of an echinococcus cyst. It is now about two and a half months since operation. The patient is back at work and feels well, but a small amount of material is draining through the sinus in the right upper quadrant.

Dr. Grantley W. Taylor: The medical service wanted to have us carry out laparotomy in this case for confirmation of the diagnosis. Peritoneoscopy had been considered as an alternative method of investigation but it was felt that this would yield less positive findings. The operative findings have been described. Many of the cysts were thick walled and necrotic and contained opaque thick purulent-like material. Others were thin walled and contained clear fluid. Two or three basinfuls of these cysts were collected and cleansed to be used for antigen. My impression was that there had been previous minor ruptures of these cysts to account for the numerous adhesions and the dispersion which was present.

I cannot agree entirely with Dr. Kranes that there is no indication for operation because sometimes large cysts of this type are ruptured by relatively minor trauma and then constitute a real abdominal emergency. In addition I think that the record perhaps fails to emphasize that the pressure symptoms were reaching the point where they caused the patient considerable trouble, not only with the function of the gastrointestinal tract but in respiration as well.

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THE *PROVIDENCE EVENING BULLETIN* LOOKS AT MEDICINE

THIRTEEN articles that appeared serially last May in the *Providence Evening Bulletin* under the ambitious title "American Medicine — Diagnosis and Prescription" have furnished a minor *cause célèbre*. This earnest effort to point out the need for reforms in medical practice cannot be dismissed lightly, for it brings to the surface certain indisputable facts. It indicates that medical service is far from perfect. It indicates the definite discontent of a considerable part of the public with the service it is receiving, and it indicates a lack of any clear understanding of the type of service that may reasonably be expected, and how it may be obtained.

The staff reporter of the *Evening Bulletin* does not alone share with the public either its discontent or its failure to see clearly the ideal relations between physicians and their patients, and how to attain them. The very many physicians of the country — the vast majority, it may be hoped — who adhere to the ancient ideals of their profession as a calling of service share also in this discontent and in the confusion that prevails regarding the best way of improving that service.

The representative of the *Evening Bulletin* as he went his way about the country, interviewing leaders in practice, in education and in public health, in an effort to present "a synthesis of some of the best thinking that is being done on how to bring adequate medical care to the people of the United States," found more questions than could be satisfactorily answered. Many of these questions have various answers with no present indications of which are the right ones. No single group or combinations of groups of persons or even that amorphous, somewhat suspect professional conglomeration known as "organized medicine" can guarantee that its own plan is the only true one regardless of its numbers or of its expertness in vociferation.

The intention of the *Evening Bulletin* was to secure thoughtful answers to such questions as these: Is there a shortage of doctors? Is "socialized medicine" the solution for our medical problems? Is there too much specialization? Why is it hard to get a doctor in an emergency? How can the cost of medical care be reduced? What are the major trends in American Medicine? Obviously the *Bulletin* is asking questions and posing problems — by no means new ones — the answers to which the Sibylline Oracles would also like to know.

Unfortunately for the interests of a fair appraisal and the securing of thoughtful answers to these vexing problems, the *Evening Bulletin's* investigator, despite the effort that he has made to reach to the heart of the matter, shows in the presentation of his theses a regrettable failure to see both sides of the issue. In discussing the difficulty that was encountered in a number of instances in finding a doctor to answer emergency calls, no true analysis of the situations was presented; twenty doctors, on a certain Sunday afternoon were unavailable or were not immediately available, and consequently "they

are to blame because the problem has been aggravated by their professional jealousy, indifference, over-specialization, sensitive pocketbooks and profound aversion to co-operative action." A strong indictment, indeed, but one suggesting so strong a bias as to weaken the rest of the witness's evidence.

The doctors of every town and every district society should be so organized that emergency calls, real or imaginary, are handled with reasonable dispatch. The need has been sounded by the American Medical Association and has been emphasized in these pages. Physicians cannot, however, keep themselves constantly on tap like hot and cold running water, as so many of the unthinking public expect of them. Certainly, organization to meet legitimate expectations should and will be put into operation wherever possible.

So go the indictments. The doctors are accused of dodging unwelcome calls (in other words, of being human); of failing in cordiality toward those elements of the population that they know intend to cheat them; of overspecializing — specialization, according to the *Bulletin*, being both the triumph and the bane of American medicine; of indifference to chronic disease and the care of the aged, in spite of the growing interest in geriatrics and the tremendous effort being exerted for the study of cancer and heart disease; of overstressing the pocketbook; of missing its chance to improve rural care and of failing to provide adequate care to millions because of rising costs.

There is no need here of offering detailed explanations or of finding excuses. Patients who have organized their own medical affairs to the point of having reliable family doctors whom they occasionally consult are much less likely to find themselves in urgent difficulties than those who use the profession only in an emergency, real or supposed, and then send their panic calls to the telephone operator or the police. Unfortunate incidents will occur; accidents will happen, and coronary vessels will occlude when no physician is immediately available. They are the chances that are taken in life.

In the practice of medicine as in other human activities, technic has outrun the present capacity of society to place scientific progress at the service of mankind. At this point while the spirit may be considered as willing, the social and economic flesh presents its weaknesses. A few workers of proved merit are responsible for the development of science; human relations are dependent on the ability of entire populations to co-operate.

It is encouraging to note the interest that the *Evening Bulletin* has shown even if it is difficult to turn all its criticisms into constructive effort.

OPERATION MUSK OX

An analysis of the many surveys and trials of rations that were conducted on the United States and Canadian troops during the war years 1941-1946* reveals that soldiers remained fit and healthy in temperate, desert, jungle, mountain or Arctic areas if they received plenty of beef, vegetables, white bread and butter, milk and coffee with sugar, fruit juice, pie and ice cream, candy and preserves, "provided they were able to avoid injury and disease"! It was also demonstrated that dry biscuits are not a good substitute for bread, that canned stewed meat and vegetables are not so good as roast beef and that the human being is unable to accustom himself to an insufficient supply of water.

The problem that the Army had to face was the reconciliation of these facts (the truth of which must have been suspected even before the surveys were conducted) with the obvious difficulty of providing the great American diet three times a day in every area in which bodies of troops had to be maintained if the main purposes of the war were to be accomplished. Here is indeed a poser toward the solution of which the nutritional scientists have little to offer. They are better able to describe what happens if the normal demands for food of the healthy young American adult are not met.

For example, men collapsed and were totally unfit for duty after three days during which their sole diet consisted of pemmican, tea and cigarets (a ration proposed by V. Stefansson because of its "lightness, compactness and easy preservability" — virtues that are also inherent in sawdust or baled hay). Jerked beef may have sustained Davy Crockett and Dr. Kane, but it won't do for Jack Armstrong.

On the other hand, participants in Operation Musk Ox, a Canadian army mobile exercise conducted in the Northwest Territories, from February

*Johnson, R. E., and Kark, R. M. *Feeding Problems in Man as Related to environment: An analysis of United States and Canadian Army ration trials and surveys, 1941-1946.* 94 pp. Quartermaster Food and Container Institute for the Armed Forces, and United States Army Medical Nutrition Laboratory, Chicago, Illinois: 1947.

to May, 1946, who were protected by vehicles, modern clothing and equipment against ill effects from the environment and were supplied by air at all times, with a wide variety of fresh food and packaged rations, experienced none of the classic diseases and hazards of Arctic travel. such as exposure, frostbite, snow blindness, psychosis, exhaustion, starvation, scurvy, edema and cold nephritis, although many of them were troubled by symptoms of carbon monoxide poisoning as a result of inhaling exhaust gases blown back into their vehicles by a strong following wind near Baker Lake, and one obese (225-pound) person lost 29 pounds between Churchill and Fort Nelson, where when seen he was in excellent health.

In desert areas, an example of misguided planning was the lining of canvas Lyster bags with a plastic to prevent evaporation. This resulted in warm water with a queer taste. By contrast, the traditional evaporating canvas desert bag produced cool, palatable water, and little was wasted. It was also learned that teaching soldiers to salt their food heavily in hot climates is preferable to giving them uncoated salt tablets, which may nauseate them.

United States troops, it is reported, will never force themselves to eat for long periods items that they dislike, even though they go hungry. This is illustrated by the tendency of the American soldier to throw away the dog biscuits in his C ration. The fact that these packages were usually at least twelve months old before they reached the consumer probably explains his reaction.

Napoleon is credited with the statement that an army marches on its stomach, and Grant with the remark that beans killed more of his soldiers than bullets. The results of these contemporary studies indicate that one must still take to heart the observations of such veteran campaigners.

TEACHER AND DOCTOR AGREE

Not infrequently articles of great interest to physicians turn up in the publications of other professions, where they usually escape the doctors' notice. A recent example is the appearance of two articles on specific reading disability in the current April and May issues of the *Independent School Bulletin*. One is by a member of the staff of the Language Clinic of the Massachusetts General Hospital,[1] and the other by a teacher at Milton Academy.[2] They are prefaced by a brief editorial note giving them the enthusiastic commendation of the principals of five well known secondary schools. The information contained in them deserves wider publicity.

The two papers are written in nontechnical language suitable for intelligent parents as well as for teachers and physicians. The first summarizes what physicians have learned about specific reading disability, and the second what an alert school can do to prevent the disastrous consequences that frequently result from failure to recognize or improper handling of children with the disability.

The authors agree that specific disability in the use of language, particularly reading, afflicts between 10 and 15 per cent of intelligent children and that, unless recognized early and handled properly, it results in scholastic failure, in a variety of behavior problems and eventually, in some cases, in misplaced and maladjusted adults. In all but the smallest communities one tenth of the pupils means a good many children, and the number for the country as a whole must run close to two and a half million.

The subject is one with which all physicians dealing with children should be familiar. Unrecognized cases are taken to ophthalmologists because teachers and parents believe that there must be something wrong with the children's eyes, to general practitioners and to pediatricians because there must be something wrong with their general health or their "glands," and to neurologists and psychiatrists because the children seem stupid or inattentive or simply mischievous at school, whereas the real trouble is in the way the victims' brains are organized. Their intelligence is normal or often superior, but their visual memory for language is so slow and faulty that they are unable to learn to read by the currently popular whole-word method. That their failure is not due to lack of attention on their part or to emotional blocking is shown by the fact that they can and do learn when they are taught phonetically with auditory and kinesthetic re-enforcement.

Koehler[2] brings out in his article that there are only ninety-four letters and letter combinations for the child taught in this way to memorize, which is a much lighter load on the visual language memory than the many hundreds of individual words that the average third grade pupil is supposed to be able to recognize by sight.

The whole word method of teaching reading in the primary grades has now been in use for many school generations. It may work well for the majority, — as physicians we are not competent to judge, — but we know from experience with our own patients as well as by the increasing complaints from the secondary schools and colleges that it fails lamentably in a large minority and is apparently responsible for much of the disability in the upper grades.

The best treatment is prevention, as outlined by Koehler,[2] which seems to succeed with most. Failing this, we have to fall back on "remedial reading," either individually or in small groups. Such tutoring is necessarily expensive and takes time. The wealthy can easily afford it, the middle class only at a sacrifice, and the majority not at all. Furthermore, there are not enough properly trained persons in the country to handle all the cases.

The only solution is to adapt the teaching of reading in the early grades to these children's capabilities. As physicians and citizens we should urge this on our local school boards and teachers.

References

1. Eustis, R. S. Specific reading disability: information for parents and teachers. *Independent School Bull.* 4: April, 1948.
2. Koehler, W. Specific reading disability: classroom view. *Independent School Bull.* 5: May, 1948.

THE COSTS OF MEDICAL CARE

As has been suspected in various professional circles, the cost of living has risen more rapidly than the fees charged by physicians for medical services. This observation is noted particularly in a study, "Comparative Increases in the Costs of Medical Care and the Costs of Living" recently published by Frank G. Dickinson, Ph.D., director of the Bureau of Medical Economic Research of the American Medical Association.

The quantity of medical care has apparently increased much more rapidly than has the number of physicians practicing; the average physician, if he is keeping up with the cost of living, is doing so by increasing the load he carries rather than the rates he charges. The public that he serves is not entirely aware of this probability. It continues to grumble at all the costs of living in an extremely complicated social and economic setup.

The quantity of physicians' services delivered in 1946 was approximately one half greater than in 1935-1939, but the number of physicians was only one seventh greater. The American people spent $5,600,000,000 for medical care in the same year, or 3.9 per cent of its total personal-consumer expenditures. This may be compared with a 4.3 per cent expenditure in 1940.

MASSACHUSETTS MEDICAL SOCIETY

DEATH

STRATTON — Harold L. Stratton, M.D., of Worcester, died on January 31. He was in his fifty-eighth year.

Dr. Stratton received his degree from Tufts College Medical School in 1939.

His widow and a sister survive.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR JUNE, 1948

Résumé

Diseases	June 1948	June 1947	Seven-Year Median
Chancroid	0	1	1*
Chicken pox	1674	1823	1282
Diphtheria	33	37	14
Dog bite	1568	1432	1318
Dysentery, bacillary	27	11	2
German measles	192	90	535
Gonorrhea	310	316	355
Granuloma inguinale	0	0	0*
Lymphogranuloma venereum	1	0	2*
Malaria	0	8	12
Measles	6569	1292	3652
Meningitis, meningococcal	4	1	16
Meningitis, Pfeiffer-bacillus	6	5	2
Meningitis, pneumococcal	0	4	4†
Meningitis, staphylococcal	0	0	0†
Meningitis, streptococcal	0	0	0†
Meningitis, other forms	3	0	1†
Meningitis, mumps	1	0	0†
Meningitis, undetermined	9	1	5†
Mumps	2034	888	966
Pneumonia, lobar	60	99	154
Poliomyelitis	3	2	1
Salmonellosis	9	7	8
Scarlet fever	915	282	791
Syphilis	203	243	391
Tuberculosis, pulmonary	259	240	264
Tuberculosis, other forms	17	25	22
Typhoid fever	5	0	3
Undulant fever	5	16	6
Whooping cough	103	508	521

*Four-year median.

†Six-year median.

Comment

Those diseases with incidence above the seven-year median are chicken pox, diphtheria, bacillary dysentery, measles, mumps, poliomyelitis, scarlet fever and typhoid fever.

Those diseases with incidence below the seven-year median are German measles, lobar pneumonia and whooping cough.

Diphtheria has had a higher incidence for June only once in the last twelve years, and measles only once since 1915. Bacillary dysentery is at the highest prevalence for June for the last nine years; mumps at the highest since 1915; and typhoid fever at the highest since 1943.

Geographic Distribution of Certain Diseases

Diphtheria was reported from: Boston, 4; Cambridge, 2; Chelsea, 2; Chicopee, 2; Foxboro, 8; Lowell, 1; Malden, 2; Medford, 1; Melrose, 1; Needham, 1; Revere, 2; Saugus, 1; Taunton, 1; Waltham, 4;Westport, 1; total, 33.

Dysentery, bacillary, was reported from: Worcester, 27, total, 27.

Encephalitis, infectious, was reported from: Lowell, 1; Lynn, 1; Millbury, 1; Quincy, 1; Saugus, 1; total, 5.

Lymphocytic choriomeningitis was reported from: Waltham, 1; total, 1.

Meningitis, meningococcal, was reported from: Boston, 1; Cambridge, 1; Fall River, 2; total, 4.

Meningitis, Pfeiffer-bacillus, was reported from: Cambridge, 1; Hatfield, 1; Holyoke, 1; Lynn, 2; Winchester, 1; total, 6.

Meningitis, other forms, was reported from: Middleboro, 1; Somerville, 1; Worcester, 1; total, 3.

Meningitis, mumps, was reported from: Walpole, 1; total, 1.

Meningitis, undetermined, was reported from: Cambridge, 3; North Adams, 1; Randolph, 1; Springfield, 1; Waltham, 2; Westwood, 1; total, 9.

Poliomyelitis was reported from: Dover, 1; Gloucester, 1; Melrose, 1; total, 3.

Salmonellosis was reported from: Brockton, 1; Chelsea, 1; Gloucester, 1; Hatfield, 1; Malden, 1; Somerville, 1; Winthrop, 2; Worcester, 1; total, 9.

Septic sore throat was reported from: Boston, 2; Cambridge, 1; Easthampton, 1; Medford, 1; Methuen, 1; North Adams, 1; total, 7.

Tetanus was reported from: Somerville, 1; Worcester, 1; total, 2.

Trachoma was reported from: Fall River, 1; total, 1.

Trichinosis was reported from: Gloucester, 1; Salem, 1; total, 2.

Typhoid fever was reported from: Boston, 3; Springfield, 2; total, 5.

Undulant fever was reported from: Lunenburg, 1; Maynard, 1; Newton, 1; Pepperell, 1; Webster, 1; total, 5.

MISCELLANY

ASSOCIATION OF SCHOOLS OF PUBLIC HEALTH

Brigadier General James S. Simmons (Retired), dean of Harvard School of Public Health, was recently elected president of the Association of Schools of Public Health at the annual meeting of the Association.

JAMES D. BRUCE MEMORIAL LECTURE AND MEDAL

The James D. Bruce Memorial Lecture in Preventive Medicine was recently delivered by Brigadier General James S. Simmons (Retired), dean of Harvard School of Public Health, at the annual convocation of the American College of Physicians in San Francisco. His subject was "The Challenge of Preventive Medicine."

General Simmons was awarded the James D. Bruce Memorial Medal for achievement in preventive medicine.

CAT-OF-NINE-TAILS DEBARRED

England's House of Lords, according to the *British Medical Journal* (June 12, 1948), debated two days and deleted the clause inserted in the Criminal Justice Bill by the House of Commons for the suspension of the death penalty for murder. Flogging was retained as a penalty, but the use of the cat-of-nine-tails was debarred.

DIRECTOR OF THORNDIKE MEMORIAL LABORATORY

Dr. William B. Castle, professor of medicine at the Harvard Medical School, has been named to succeed Dr. George R. Minot as director of the Thorndike Memorial Laboratory of the Boston City Hospital, where he has been associate director since 1932.

With Dr. Castle's appointment, Dr. Minot, Nobel Prize winner in 1934, retires from his joint positions as professor of medicine at the Harvard Medical School and director of the Thorndike Memorial Laboratory.

Awarded the Nobel Prize in Medicine and Physiology in 1934 with Dr. William P. Murphy, of Boston, and Dr. George H. Whipple, of Rochester, New York, for discoveries in the treatment of anemias, Dr. Minot has devoted over thirty years to medical research teaching and has made important contributions to the subjects of anemia, abnormal bleeding and the treatment of leukemia by x-ray.

Dr. Castle has received honorary degrees from Yale University and from the University of Utrecht, Holland. He is also the recipient of the John Phillips Memorial Award of the American College of Physicians, the William Proctor, Jr., International Award for Distinguished Service in Promoting Health, and the Walter Reed Medal of the American Society of Tropical Medicine.

ODONTOLOGIC CHARACTERISTICS OF LONG-TAILED PRIMATES

New World monkeys, according to Sir Frank Colyer, (*Brit. M. J.* **1**:1202, 1948) differ from Old World monkeys in various anatomic particulars, especially various odontologic characteristics. The green monkeys of St. Kitts, however, exhibit the greatest capacity for dental variations. The incidence of supernumerary teeth, abnormal positions, rotations and inward displacements is three or four times as great in this species as in comparable groups of African monkeys.

STUDENTS OF ADMINISTRATION PLACED

At the completion of the first year of Yale University's course in hospital administration, 8 students found immediate placement. Of these, 1 remained at the Grace–New Haven Community Hospital; 5 found positions in Boston; 1 accepted an administrative internship in Rochester, N. Y.; and 1 will fill a similar position in Valhalla, N. Y.

CORRESPONDENCE

IN CORPORE SANO

To the Editor: Congratulations to Professor Koon on his article in the *Boston Traveler* on June 11, 1948. The last paragraph says, "Bark beetles which carry the fungus of the Dutch elm disease will not breed in healthy living tree tissue. Keep your elms healthy by feeding them."

The organic gardeners have proved for years that healthy plants are practically free from diseases and bugs. Many people, including some doctors, are of the belief that a good part of the increase of cancer is due to poor food. Much of this poor food is due to an excessive use of commercial fertilizers. The organic method, with no commercial fertilizers, dusts or sprays and plenty of humus in the soil, produces vegetables that make healthier people and less sickness. The organic gardeners are called zealots or faddists by some. It is the opinion of many that those opposed to organic gardening are either jealous or are connected in some way with the all-powerful chemical companies. We would not go back to the old chemical method if the chemical companies offered us a life pension!

George H. Crosbie
An organic gardener

North Scituate
Massachusetts

NOTICES

ANNOUNCEMENTS

Dr. Albert Aranson announces the opening of his office at 73 Deering Street, Portland, Maine, for the practice of internal medicine and chest diseases.

Dr. William H. Horwitz announces the removal of his office to 1093 Beacon Street, Brookline, for the practice of pediatrics and pediatric allergy.

(*Notices concluded on page ix*)